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# Archives OF SURGERY

FEBRUARY 1956 VOLUME 72 NUMBER 2

A Glimpse of Russian Surgery — *F. Henry Ellis Jr.*

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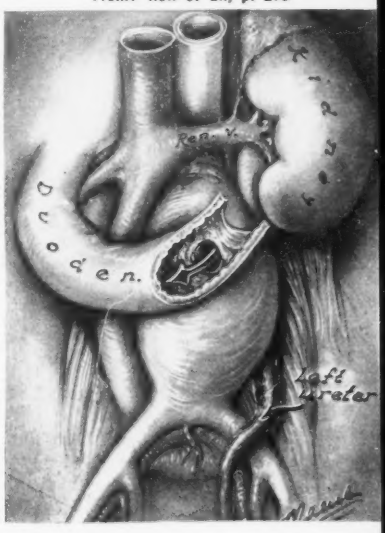


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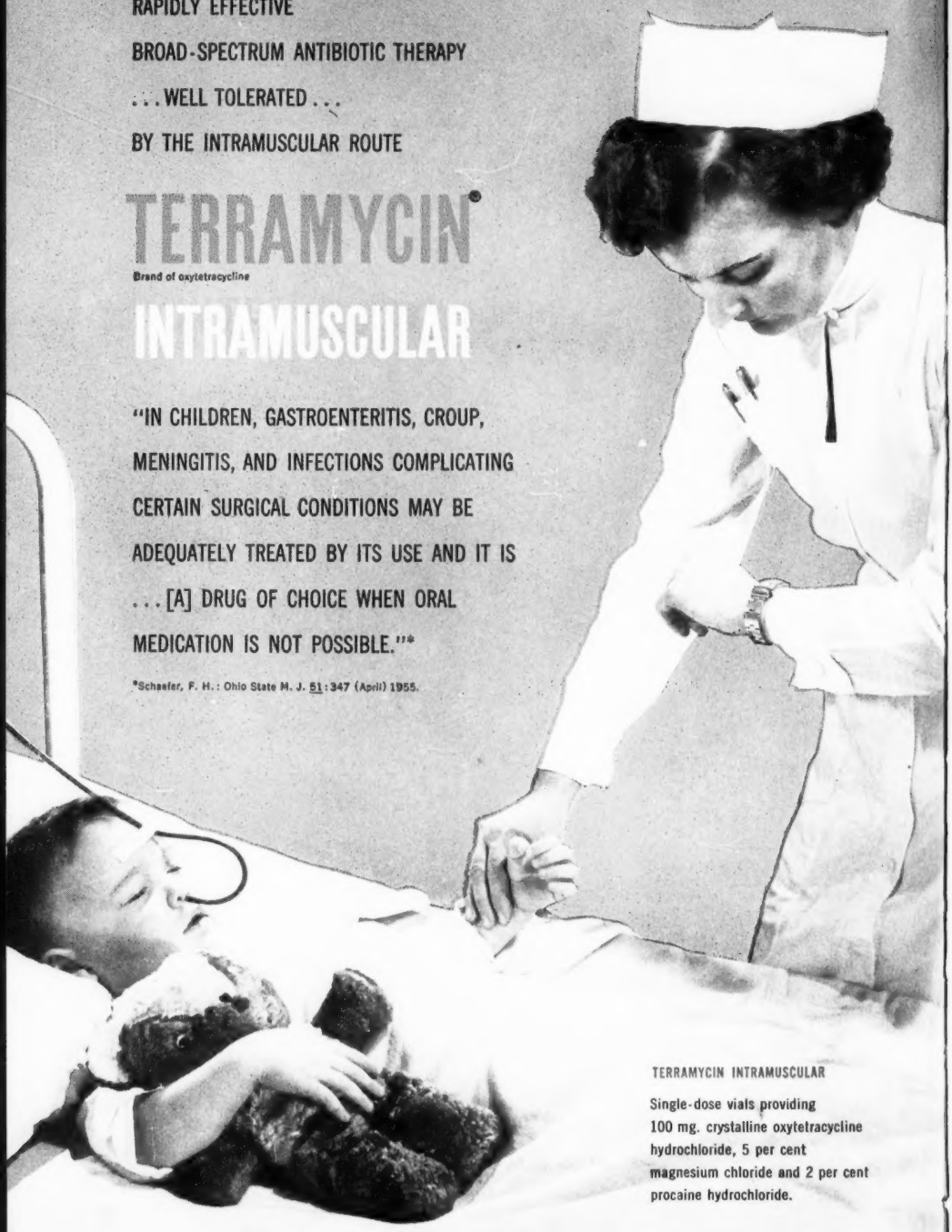

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# ARCHIVES OF SURGERY

**Also the Official Publication of the Central Surgical Association and the Western Surgical Association**

VOLUME 72 — FEBRUARY 1956 — NUMBER 2



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**The Journal of the American Medical Association.** Weekly. Annual Subscription Price: Domestic, $15.00. Canadian, $17.00. Foreign, $21.50. Price to students, interns and residents: $9.00 in U. S. & possessions.

**Quarterly Cumulative Index Medicus.** Issued Twice a Year. Subscription Price, Calendar Year, $25.00.

Checks, money orders, and drafts should be made payable to the American Medical Association, 535 North Dearborn Street, Chicago 10.



Published Monthly by

AMERICAN MEDICAL ASSOCIATION

535 NORTH DEARBORN STREET • CHICAGO 10, ILLINOIS

Entered as Second Class Matter July 10, 1925, at the Postoffice at Chicago, Under the Act of March 3, 1879. Annual Subscription, $14.00

A.M.A. Archives OF SURGERY

# A Glimpse of Russian Surgery

F. HENRY ELLIS Jr., M.D., Rochester, Minn.

*Russian surgeons have attended and have presented scientific papers at the meetings of the International Society of Surgery in Paris, in 1951, and in Copenhagen, in 1955.*

*A scientific meeting was held in Russia within the last year that was participated in by Russian surgeons and their invited guests, of whom two were invited from each of many countries. According to one guest, this was the first time such a meeting of Russian surgeons had been held since World War II.*

*Visits to Russian hospitals have been made in recent years by foreign physicians; the report of one, namely Dr. Fox, editor of "The Lancet," was published in that journal. Few Americans have had an opportunity to gain knowledge of Russian scientific advances in medicine and surgery until this summer, when Major Paul Schafer of the U. S. Army Medical Corps was invited to Russia by the Russian Ministry of Health. Two other American surgeons, Dr. F. Henry Ellis Jr., a surgeon of the Mayo Clinic and Mayo Foundation, and Dr. G. Arnold Stevens, Clinical Professor of Surgery at the University of California, Los Angeles, made individual requests for visas to visit Russian medical centers. These visas were granted, and the report of Dr. Ellis will be found in this issue. I am sure the information in it will prove of considerable interest to American surgeons; we hope that Major Schafer will be able to send us a report of his observations in the not-too-distant future.*

WALTMAN WALTERS, M.D.

Since the end of World War II, little information has been available in this country regarding Russian medical standards and medical practices. Some Russian journals, it is true, have been regularly received by certain of our libraries, but because of language difficulties the information contained therein has not been widely disseminated. Visits to Russian hospitals have been made in recent years by foreign physicians. Only last fall a group of English physicians spent several weeks on a conducted tour of various Russian medical facilities.[1] Although the experiences and impressions of these English practitioners are known to many of their colleagues, few Americans have been made aware of the information they obtained. In fact, until this summer, when Major Paul Schafer, of the United States Army Medical Corps, was invited to Russia by the Russian Ministry of Health, no American physician had visited Russian hospitals since the end of World War II, as far as I know.

Because of the general lack of knowledge of Russian medicine, my experiences during a two-week visit with Russian surgeons in Moscow and Leningrad may be of interest. I must preface these brief comments, however, by emphasizing several things. My tour was a limited one, encompassing as it did only the major medical centers in two of Russia's major cities. For this reason I cannot speak for medical conditions in other parts of the U. S. S. R. Furthermore, my contacts were, of necessity, made through an interpreter. Information so gained is subject to the obvious errors and misunderstandings related to translation problems.

The fact that my visit was an unofficial one, for which the Russians apparently were

Submitted for publication Dec. 15, 1955.

Section of Surgery, Mayo Clinic and Mayo Foundation. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

unprepared, makes me believe that the things I saw were not necessarily carefully planned in advance so as to give a prearranged impression. My visit took place during the last two weeks of September. A tourist visa was granted me six months after I had made application to the Russian Embassy in Washington. Contacts with Russian physicians were not made in advance, but were made after my arrival in Russia through Intourist, the Russian tourist agency. However, letters of introduction to Professor Petrovski, of Moscow, who is a vice president of the International Surgical Society, were graciously provided me by Drs. Evarts Graham and Walter G. Maddock. In addition, Dr. Fox, editor-in-chief of *The Lancet*, a member of last year's British delegation to the U. S. S. R., was kind enough to advise me about the several hospitals that I should visit.

### GENERAL ASPECTS OF MEDICINE IN THE U. S. S. R.

Since the revolution in 1917, Russian medicine has been socialized, but, surprisingly enough, private practice is not prohibited. However, it is rare for a Russian to pay his physician for services rendered. There are approximately 300,000 practicing physicians in the U. S. S. R., about 50% to 60% of them being women. The high ratio of women physicians may be related to two things. Undoubtedly, many more women studied medicine during the second World War, when men were needed for the armed forces. Furthermore, the majority of Russian women seem to be employed, and professional opportunities are offered them more freely than in our country. Yet, in spite of the great number of women physicians, there were few women surgeons in high positions in the hospitals I visited.

General policies regarding medical care throughout the Soviet Union are determined by the Ministry of Health. There are subsidiary ministries of health in the various republics which make up the U. S. S. R. The impression gained, however, was that these were not autonomous groups, but relied on Moscow for their directives. The physicians themselves are active in the Ministry of Health, many of the high positions being held by physicians who at one time or another have been active in the clinical practice of medicine.

A great deal of emphasis is placed on prophylactic medicine, and routine examinations frequently are made of workers in the various industrial and farm groups throughout the country. Mass thoracic x-ray surveys are made, and I was told that the tuberculosis rate, while showing a slight increase during and after the second World War, is not high. Heart disease and cancer are, as in our country, the chief causes of death. Cancer of the stomach remains, however, the leading cause of those deaths caused by cancer, and no particular increase in the incidence of cancer of the lung has been noted. No relationship between smoking and cancer of the lung has been found, although cigarettes seemed plentiful and were freely used in the cities I visited. It may be of interest that the cigarettes are constructed differently from ours. Two-thirds of the length of a cigarette is composed of a hollow tube; only about a third of the cigarette contains tobacco.

In a city such as Moscow, medical care is provided by a system of "polyclinics" and general hospitals. The polyclinics are similar to dispensaries, and serve specific geographical areas of the city. Patients either go to the polyclinic, where outpatient treatment, diagnosis, and minor surgery are carried out, or they are visited at home by physicians staffing these polyclinics. The patients are sent from the polyclinic to the hospital serving that district when hospitalization is required. As a general rule, hospital and polyclinic physicians are not interchangeable, although one hospital I visited helped to staff its referring polyclinic on a rotating basis. Whereas in the hospital all medical care is free, the patient is required to buy most of his own drugs, when possible, when he is being treated at the polyclinic.

### MEDICAL EDUCATION

A Russian boy or girl enters medical school after a competitive examination fol-

lowing graduation from the equivalent of our high school, usually at the age of 17 or 18 years. There are now about 75 medical schools in the U. S. S. R. The classes in some of these institutions are very large, around 600 to 700. There are three faculties in many of the medical schools, therapeutics, pediatrics, and hygiene, and the graduating student gets his doctor's degree with a notation on his diploma as to the faculty under which he studied. The faculty of therapeutics trains students for a career in medicine, surgery, and the subspecialties. A course in pediatrics emphasizes maternal and child care, whereas hygiene stresses public health and epidemiology. The courses in the three faculties have many similarities, however, and transfers from one to another occur. During the first three years of the six-year course, instruction is given in the basic sciences and a foreign language is studied. I have read that there is also some political indoctrination. The student enters the hospital in the fourth year, and during the last year he concentrates on what is to be his special field of interest. During the student's period of training, he receives a stipend from the state of 300 to 400 rubles a month at the start, increasing to 600 rubles later on. A student's stipend may be lowered or taken away if he fails in his courses. On the other hand, bonuses and increases in stipends are given to the more brilliant students. After graduation, a student is assigned to practice somewhere in the U. S. S. R. for a period of about three years. Depending on the desirability of the location, he may receive more or less money. The student's wishes regarding his place of practice are considered, I was told, but I suspect there is little hesitation on the part of the authorities to assign physicians to areas where they are needed. There is no compulsory military service for physicians except in time of war, since the Russian armed forces have their own medical schools, from which their medical requirements are adequately filled. I was not told on what basis students are originally assigned to these military medical schools.

### SURGICAL TRAINING

A physician who wishes to become a surgeon returns to a medical center in one of the larger cities after his three years of practice in an outlying district. In special cases an outstanding student may go directly from medical school into a hospital for postgraduate training. He works for three years in the hospital as a resident, at the end of which time he is qualified to practice surgery independently. In one of the hospitals I visited, I was told that a surgeon in his first year of training carries out such procedures as appendectomies, herniorrhaphies, reduction of fractures, and decompression for brain injuries. In the second year he progresses to intestinal anastomoses, mastectomies, thyroidectomies, and cholecystectomies. In his last year such procedures as gastrectomy, resections of the colon, lobectomy, and pneumonectomy are performed. Specialization is not so clearly defined as in this country, and much of the thoracic surgery is done by general surgeons, there being no special training program for those interested in this field.

Having completed the three-year hospital training period, a surgeon may seek extra training if he is qualified, and if he wishes to continue at one of the larger centers in an academic position. A further training period of three years is required for such a course, during which time a thesis must be written, based on experimental work, and defended, much as a doctor of philosophy thesis is written and defended in this country. In addition to this formal course of surgical training, practicing surgeons may periodically return to the larger teaching centers for refresher courses in surgery. These courses may last from three to nine months. I encountered several such surgeons during my visit to Moscow hospitals.

The surgeon's position in the over-all social scale of the Russian world is an extremely high one. It is hard to compare Russian salaries with ours in terms of dollars and cents because of the artificial exchange rate, for one reason, and because a Russian,

although well paid, may find little to buy in government-owned stores, but may be forced to buy at exorbitant prices in the so-called free market. Whereas the average worker in Russia receives 650 to 750 rubles a month ($162.50 to $187.50) at the current exchange (of 4 rubles for $1.00), the average young surgeon makes about 2000 rubles a month, and a professor of surgery may make 6000 to 8000 rubles a month. A professor who is also head of a department may be paid considerably more, particularly if he has written a textbook which is widely used. Royalties from such sales are paid to the author, and after his death they continue to be paid to his children. Such was the situation in the case of Prof. Sergei Yudin, the internationally known esophageal surgeon of the Sklifosovski Institute in Moscow. Although I never visited a Russian surgeon's home, Dr. Pietro Valdoni, of Rome, who did, described a typical professor's home to me. A man in such a position may have a five-room home in the country, a servant, and a car and driver supplied by the state. I was astonished to learn that physicians and nurses are required to work only six hours a day for a six-day week. Extra pay is received for overtime work.

### HOSPITAL FACILITIES

In Moscow two of the important surgical clinics, the Spasokowkorski Institute and the Sklifosovski Institute, were being remodeled, and a new 250-bed surgical unit for thoracic and cardiovascular patients was being constructed. The average hospital has a drab and dull exterior, needing paint and replastering. I visited no modern hospital buildings comparable to some of the magnificent medical buildings to which we are accustomed in this country. Little effort was made to maintain attractive grounds. The interiors, however, were well kept up, light, and cheerful. Potted foliage of various sorts adorned the corridors, and an obvious effort was made to keep up the patients' spirits.

In each hospital I visited, the hospital arrangement was somewhat confusing. With the exception of the Botkin Hospital (a large city hospital of about 2400 beds), the hospitals themselves seemed to have no names. Rather, a group of hospital buildings would contain various institutes. Thus the Petrovski Institute and the Spasokowkorski Institute were small surgical units within a large hospital that contained other institutes as well. Members of these institutes might be members also of the First or Second Moscow Medical Institute, which would have many small institutes in other hospitals under its jurisdiction.

There was no shortage of personnel, both professional and nonprofessional. There were many physicians, nurses, nurses' aids, and other workers in evidence at all times. Nursing apparently is a favored profession for young women, I was told by my interpreter. There are two nursing courses, one of three years' and one of two years' duration. The two-year course equips a girl for less exacting duties, comparable, I suppose, to those of our vocational nurses. The three-year course, on the other hand, provides a more extensive nursing training, including some rudiments of medicine, for in some outlying areas a nurse may be called upon to perform some of the duties of a physician.

In one surgical unit, the Spasokowkorski Institute headed by Professor Bakulev, a cardiac surgeon, 70 surgeons, including the residents and others in training, were on the staff; the bed capacity was 325 beds.

Laboratory facilities seemed adequate. Facilities for frozen-section preparation of surgical tissue were available in at least one of the hospitals I visited. X-ray equipment was, on the whole, rather inferior to ours, although in one hospital I saw a Swedish angiocardiography machine capable of taking 12 pictures a second. Electrocardiography laboratories and chemistry laboratories were available, and in addition I saw facilities for ballistocardiography, Van Slyke gas analysis, and cardiac catheterization. The Russians have an ear oximeter of their own manufacture which I saw in use during a heart operation.

The operating rooms were clean and spacious, some of the rooms accommodating two operating tables. Most of the equipment was of Russian make, although I did see an American operating-room light and sterilizer in one hospital. Gas machines for anesthesia did not seem to be of very high quality. Several operating rooms were equipped with conduits for anesthetic gas running under the floor from supplies located in a separate room. It was common practice for postoperative recovery rooms to be located adjacent to the operating-room suites. All postoperative patients were kept in these rooms for several days after operation before being returned to their wards. The wards usually consisted of six to eight beds, and there seemed to be no crowding of patients. A few smaller rooms also were available. The patients appeared to be well cared for, and were provided with suitable hospital gowns and robes.

### CONDUCT OF SURGICAL PROCEDURES

There was some variation in the standards of the surgical procedures that I witnessed. Whereas in one hospital the sterile technique was entirely acceptable according to our standards, in another the attention to sterile technique was only cursory. The senior surgeons whom I saw operate were extremely skillful, and operated rapidly. They were, on the whole, less meticulous than American surgeons, and their technique did not involve particular care for the tissues. Hemostasis was rather slipshod, and heavy gut, with large bites of tissue, was commonly employed. Local anesthesia and spinal anesthesia are widely used. It is not uncommon for an entire transthoracic esophagogastrectomy to be carried out with the patient under local anesthesia. One surgeon, I was told, used local anesthesia for mitral commissurotomies.

Fluids for intravenous use were not available in sealed bottles, as in this country, but were poured from flasks into a drip apparatus connected to the patient. Blood was readily available and was used freely. Blood from cadavers is used in about three-fourths of the transfusions carried out at the Sklifosovski Institute. Professor Yudin introduced and made acceptable the use of blood from cadavers at the hospital in the 1930's. This type of blood also is used at other hospitals, but not as extensively. The blood is drawn aseptically six hours after death of the patient and is kept at 4 C for no more than 30 days. I was told that 20 tons of such blood had been used at the Sklifosovski Institute and that those who administer it are entirely satisfied with its use. At another hospital donor blood is preferred when available.

The operative procedures carried out differ very little from those to which we are accustomed. During my stay in Russia, I witnessed an aortic pulmonary anastomosis done for tetralogy of Fallot, carried out under intratracheal anesthesia and beautifully performed by Professor Michalkin, of the Spasokowkorski Institute. I also saw an appendectomy, an anterior resection for carcinoma of the rectosigmoid, and an esophagogastrostomy through an abdominal approach for what proved to be an inoperable carcinoma of the cardia. A typical operating schedule for one morning at one of the hospitals was as follows: two appendectomies, two cancers of the rectum, and a nailing of a fractured femur. Records of the operative procedures carried out during the preceding years were shown me in one hospital; they included most of the procedures commonly in use in this country. In the wards of the hospitals I saw patients who had undergone thyroidectomies, gastric resections, esophagogastrectomies, mitral commissurotomies, pneumonectomies, and cholecystectomies. For the most part, their convalescence seemed satisfactory and the patients appeared to be well cared for. Antibiotic agents, such as penicillin, streptomycin, and chlortetracycline (Aureomycin), were available, but they did not seem to be used indiscriminately.

The wide use of local anesthesia by Russian surgeons has caused much comment. The following facts may be a partial explanation. Local anesthesia has always been used more freely on the continent than in this country. Unquestionably, well-trained anes-

thesiologists are not numerous in Russia. The character of the Russian person is responsible to some extent, I am sure. He appears to be stoical and accustomed to suffering and, I am sure, can tolerate physical stresses which patients in this country never could withstand.

I was fortunate enough to receive a patient's first-hand account of surgical care in a Moscow hospital from Mr. James Lionel Menning, sports editor of a large London daily newspaper. Mr. Menning, who had been sent to Russia to write a story on Russian sports, had the misfortune to be attacked by appendicitis the day after his arrival in Moscow. Being a new correspondent and knowing no one, he was considerably discomfited. He at length was forced to call for medical assistance through the agency of the hotel. A Russian physician visited him in his room within a half-hour, diagnosed his condition, and recommended immediate appendectomy. After a hurried consultation with the British Embassy, he was taken to the hospital, where he was prepared for operation. The operation was performed under local anesthesia by a Mongolian whose appearance over the mask did little to reassure the extremely anxious patient. Although he found the operative procedure uncomfortable, it was not unbearable and was completed within about 40 minutes. The Mongolian apparently was in training, for a senior surgeon was present, and, although he was not scrubbed, he did much to allay the patient's fears.

The postoperative care was excellent. The patient was provided with a private room. There were many nurses available, and all of them were extremely solicitous. The food was good, and everything possible apparently was done to make him comfortable. He was allowed to be out of bed on the third postoperative day. The stitches were removed on the 6th day, and he was back at his hotel on the 10th day, none the worse for his experience. The British medical attaché, it is interesting to note, was not allowed to accompany him to the hospital or to visit him while he was there. In this regard it is interesting that Dr. Herbert, of the United States Air Force, who is medical attaché at the American Embassy, told me that for many months he had tried unsuccessfully to visit Moscow hospitals. He finally made such a visit with Major Schafer. At no time, however, was I able to obtain permission for him to accompany me on my visits to the hospitals.

### SPECIAL SURGICAL PROBLEMS

During this short visit I was able to cover superficially only a small fraction of the whole field of surgery. The following data are some of the bits of information I was able to obtain.

*Thoracic Surgery.*—Surgery for tuberculosis has developed rapidly. Pulmonary resection is the commonest surgical procedure performed, about 25% of tuberculous patients being treated by some form of surgery. Pneumothorax is still widely used, and thoracoplasty is employed more than it is in this country. Pulmonary resection is not advocated for so-called residual necrotic foci.

Esophageal surgery is rather advanced, as a result primarily of the excellent work of Yudin. Lye strictures of the esophagus are still common in Russia, and at the Sklifosovski Institute, a surgical unit of more than 1000 beds, more than 700 esophageal reconstruction operations have been performed. I saw 8 or 10 such patients in the wards of this hospital in various stages of reconstruction. The procedures used vary considerably. The jejunum is used for reconstruction in the majority of cases, an isolated limb being fashioned and anastomosed to the esophagus above and the stomach below. If necessary, the jejunum is anastomosed to the esophagus in the neck. In such cases the jejunum is brought superiorly through the anterior mediastinum retrosternally. The transpleural approach also is used in suitable cases. In a few instances an attempt has been made to support the blood supply of this isolated jejunal limb by anastomosing to its arterial branches the internal mammary artery or

right gastroepiploic artery. Twenty-three such procedures have been done.

There was some variation in the approach of different surgeons to the problem of cancer of the lung. Most thoracic surgeons advocate pneumonectomy. At one hospital, however, patients are no longer operated upon but are treated with roentgen rays. Another surgeon for several years has been carrying out rather radical pneumonectomy, with intrapericardial dissection in selected cases.

*Cardiovascular Surgery.*—It is my impression that, on the whole, progress in cardiovascular surgery has not reached the stage of development that it has in this country. The preoperative evaluation of cardiac patients depends primarily on angiocardiography, although cardiac catheterization techniques also are employed. Operations for patent ductus arteriosus, coarctation of the aorta, and procedures such as aortic pulmonary anastomosis for tetralogy of Fallot, pericardectomy, and mitral commissurotomy, are performed regularly in centers which specialize in cardiac surgery. At the Spasokowkorski Institute cardiac surgery is carried out in considerable volume. I saw several patients who had undergone mitral commissurotomy, and others who had undergone other cardiac procedures, convalescing satisfactorily at this hospital. I was told that the mitral valve usually is opened by blunt dissection and that the knife is seldom employed. The incidence of cerebral embolism in such cases was quoted to me as 2%. In some cases patent ductus arteriosus is treated by ligation and in others by division. Aortic stenosis and insufficiency are not treated surgically. Operations for septal defects are not well advanced, most of the work being of an experimental nature, although a few patients with such defects had been treated in Leningrad at Kuprianov's clinic by atrioseptopexy. Hypothermia with caval occlusion had been employed for open operations on the heart at this hospital in several cases of tetralogy of Fallot in which infundibulectomy had been carried out. The ventricular septal defect was not closed in these cases. Although aortic grafts had been used in some cases of coarctation of the aorta, resection and grafting for aneurysms and occlusion of the abdominal aorta had not been done, so far as I could determine. Grafting procedures had, however, been carried out in cases of trauma and arteriovenous fistulas of peripheral vessels. Blood-vessel grafts were preserved in a liquid medium and were not freeze-dried. At the Sklifosovski Institute an ingenious instrument has been devised by Professor Androsov, for which he recently was awarded the Stalin Prize. This instrument is used to anastomose blood vessels with a circumferential series of fine tantalum staples. The demonstration that I saw carried out on the blood vessel of a cadaver was impressive. In actual use the instrument might be somewhat cumbersome. Similar ingenious stapling instruments are employed for ligation of vessels and closure of the bronchial stump.

*Gastrointestinal Surgery.* — The surgical treatment of duodenal ulcer remains subtotal gastrectomy. It was difficult for me to be sure in what percentage of cases such treatment is employed. Perforated duodenal ulcers are treated by subtotal gastrectomy in many cases, when the patient's condition permits. Gastroenterostomy and vagotomy for duodenal ulcer do not seem to enjoy much favor. Total gastrectomy for carcinoma of the stomach is done only when the lesions are very extensive.

At one hospital that I visited, sphincterotomy was heartily endorsed as a good form of treatment for chronic pancreatitis. Operative cholangiography also was employed in this hospital, although other surgeons with whom I talked said they do not employ this procedure.

Splenorenal and portacaval shunts were being performed for bleeding esophageal varices in some institutions, while hepatic artery ligation was being done for the same condition at another institution.

*Miscellaneous.*—Thyroidectomies are routinely carried out with the patients under the influence of local anesthesia. Radioactive iodine is available and is used in the treatment of some patients with exophthalmic

goiter. The method of selection of patients for treatment by this method was not clear to me. A variety of radioactive substances apparently is available, for both the treatment and investigation of diseases.

Early ambulation is not stressed in Russia to the extent that it is in this country, although I was told that greater emphasis on this had been made in the past. Heparin and bishydroxycoumarin (Dicumarol) are used in the treatment of thromboembolism, but are not given prophylactically to any postoperative patient.

In general, I would say that the surgical approach to cancer in the U. S. S. R. tends to be less radical than it is in this country. Procedures such as radical exenteration for pelvic malignant processes, dissections of internal mammary nodes for mammary carcinoma, and hemimandibulectomy and radical neck dissection for cancer of the head and neck are not widely practiced, if at all, so far as I could tell.

### RESEARCH PROGRAM

An active research program apparently is being carried out in Moscow. The research laboratories, however, seem to be located in separate buildings quite apart from the hospitals. Transplantation of organs and portions of the anatomy from one animal to another is being studied extensively. The experimental aspects of various prosthetic agents for use as vascular grafts are being studied. Hypothermia is being investigated as a means of allowing open cardiotomy for repair of intracardiac defects. I was told by several surgeons that an experimental heart-lung machine for maintaining extracorporeal circulation during open cardiotomy is under investigation in Moscow. Although I tried to see this machine, I was not allowed to do so. I could not determine whether the machine had been used successfully on experimental animals. I was told, however, that difficulties are being experienced with the oxygenator.

I was fortunate enough to visit an experimental physiology laboratory of the Academy of Medical Sciences, headed by Professor Negovsky. This laboratory is located on the second and third floors of one of the oldest buildings in Moscow, far removed from any other scientific activity. Seventeen physicians worked in these laboratories, some full time and some part time. Most of the physicians were women. Complete laboratory facilities were available here for chemical analysis of body fluids and pathologic examination of tissues. The chief work under way at this laboratory was concerned with studies on the "terminal states," hypothermia and defibrillation. I witnessed an experiment in which a dog was brought back to life after having been "dead" for five minutes. Obviously, our definition of death and that of the Russians are at variance. This experiment consisted of bleeding a dog until corneal reflexes were absent and blood pressure could not be measured, and then restoring these functions by the reinfusion of blood. An electrocardiogram taken during the procedure showed persistence of cardiac activity, however, and the experiment actually consisted of the alleviation of exsanguinating shock, by means of replacement of blood.

Defibrillation by direct current was being investigated in this laboratory, a current of 1000 to 3000 volts at 8 to 10 amperes applied for 0.01 second being used. Defibrillation was accomplished in a dog by the application of an electrode to the surface of the closed thorax of the animal.

### GENERAL IMPRESSIONS

The kindness and humanity exhibited by the Russian surgeons toward their patients were a surprise to me. In spite of the fact that the Russian physician is responsible primarily to the state, the physician-patient relationship seems to be salutary.

Although I was not prepared for the freedom with which I was shown the hospitals I visited and was gratified by the apparent candor and honesty of the Russian physicians whom I met, total freedom of movement was by no means available. Access to the hospitals required several days, during which time I am sure the Intourist agent at the hotel

awaited instructions from some higher echelon, perhaps the Ministry of Health. Telephone books are not generally available in Moscow, and so, even had I been more familiar with the language, I would not have been able to telephone to a Russian physician. More disconcerting than anything else, I think, was the fact that not one of the Russian physicians whom I met would communicate with a colleague of his and arrange a meeting with him for me. It was always necessary to develop these contacts through the Intourist agency in the hotel. As I left Moscow for Leningrad, I tried repeatedly and unsuccessfully to get a letter of introduction to Professor Kuprianov's clinic, where open cardiotomy is being performed under hypothermia and inflow stasis. I visited this clinic only after the Intourist agency in Leningrad had made the arrangements.

All the Russian surgeons were extremely hospitable and gave liberally of their time. They were intelligent and well informed, and their questions reflected good training and background. It was obvious that our medical literature is readily available to the Russian surgeon in the larger institutions. It was not uncommon to see our journals on the professors' desks and our textbooks in their libraries.

The intense chauvinism which I expected was rarely apparent. Russian physicians and surgeons pointed with pride to Russian-made instruments but at no time claimed Russian priority for any discovery, operative procedure, or invention. On the contrary, credit was given where credit was due, and most of the surgeons were quite frank in admitting they had not kept pace with American progress in cardiovascular surgery.

On the whole, the men I met struck me as similar to men of science the world over. They were interested in their specialty, interested in their patients, and interested in sharing knowledge and experiences with a visiting scientist, regardless of his political affiliations. They even went further and repeatedly expressed the wish, which seemed sincere, that closer contacts between Russian and American surgeons would be possible in the future.

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# Ligation of the Inferior Vena Cava After Nephrectomy

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and
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We believe the case reported here to be the first of its kind reported, and, since the indication involved must certainly recur, we believe it to represent an addition to the surgeon's armamentarium.

Virchow,* between 1846 and 1856, suggested that embolism was related to phlebitis of the legs.

Kocher † first ligated an inferior vena cava for surgical trauma in 1883. Billroth repeated the procedure in 1885. Both operations were fatal. The first success with ligation for trauma is credited to Bottini, reported by Costa in 1921, in the *Italian Journal of Surgery*. Trendelenberg ligated two venae cavae in 1906 and a third in 1910, all for thrombophlebitis with septicemia. Pfaff [3] stressed the fact, in 1926, that it was safer to ligate the injured inferior vena cava below the level of the renal veins than to attempt to repair it.

The concept of ligation for the prevention of emboli was suggested by Homans [4] in 1934. It is chiefly since Homans' work that caval ligation has played a prominent role in the treatment of thromboembolic disease. Impetus has been added by Homans' observation of the bilateral nature of the disease and the frequency of emboli from the contralateral side when ligation of a thigh or pelvic vein is carried out. Impetus has also been added by the excellent and growing work of Collins [5] and his associates, of New Orleans.

The indications for caval ligation have expanded and now can be enumerated by combining the indications of Homans with those of Payne.[6] They are as follows: 1. trauma, operative or otherwise; 2. therapy for active, acute, extending vein disease or its sequelae; 3. last stage procedure to prevent pulmonary embolism (we no longer save it for the last stage); 4. prophylactic measure at the time of abdominal exploration for another condition, and 5. phlegmasia cerulea dolens (blue phlebitis of Gregoire [7]).

In 1950, we ligated an inferior vena cava in a situation not described in the literature and one that must recur from time to time. The patient survived and is well. This case is presented to increase the surgeon's armamentarium and as a new indication for ligation.

## REPORT OF A CASE

The patient, a white woman 69 years of age, entered St. Joseph's Hospital, Fort Wayne, Ind., April 10, 1950. She had undergone a right-sided nephrectomy in the same institution 47 days before, on Feb. 22, 1950. The pathological diagnosis was chronic pyelonephritis and renal lithiasis. Convalescence had been normal, and there were 17 days of hospitalization. Her blood cell count on previous admission was normal throughout, and blood pressure had averaged 180 systolic and 100 diastolic.

The patient pursued a normal convalescence at home until April 8, 1950, forty-five days after surgery, when, at 9 o'clock in the evening, she experienced sharp pain in the right side of the abdomen, radiating to the shoulder, neck, and right chest. She vomited two hours later. After that she had continuous pain of varying severity over her entire

Received for publication April 7, 1955.

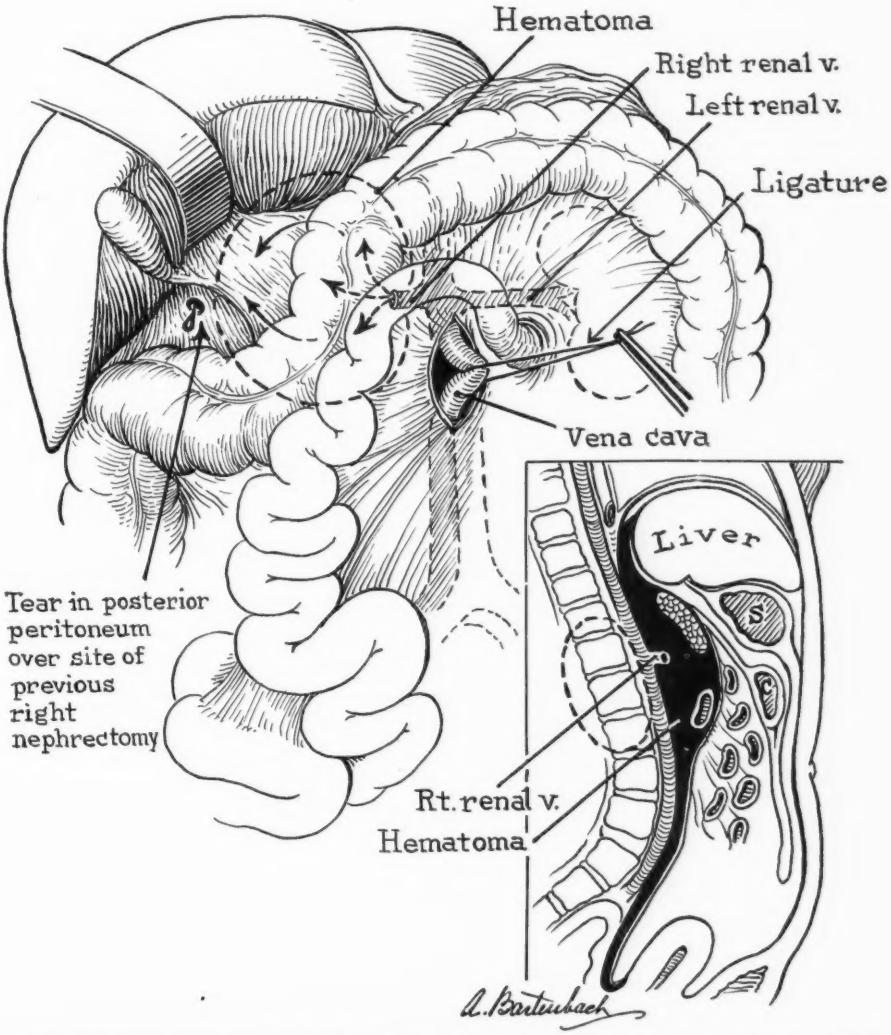
Specialist certified by the American Board of Surgery (Drs. Bolman and Lloyd); fellow, American College of Surgeons [Western Surgical Association, 1955 (Dr. Bolman)].

* Cited by Welch.[1]

† Cited by Moses.[2]

abdomen. She continued to vomit during this time. Her bowels moved with enemata, but there was no relief. She was brought back to St. Joseph's Hospital at 7 p. m., April 10, 1950, forty-seven days after her surgery. We were asked to see her shortly thereafter.

Examination revealed an extremely pale and apprehensive elderly white woman. Her abdomen was diffusely tender, and there was a doughy type of rigidity. Bowel sounds were absent. Rebound tenderness was everywhere marked. Blood pressure was 90 systolic and 70 diastolic; pulse rate was 100, and respiration rate was 26. The patient made repeated and continuous fruitless attempts to vomit. The chest was clear. Reflexes were equal and present. Urea nitrogen was 15.2 mg. per 100 cc. Red blood cell count was 2,800,000, with 8.0 gm. of hemoglobin. White blood cell count was 16,400, with 87% polymorphonuclear leucocytes and 13% immature forms. Platelets were 185,000 per cubic millimeter. Urine was normal. Scout film of the abdomen was uninformative. The tentative diagnosis was intra-ab-




The main figure shows the situation found at laparotomy. The ligature has already been placed under the vena cava. The inset shows the extent of the hematoma as viewed in sagittal section.

dominal hemorrhage, origin undetermined. It was difficult to relate the bleeding to the operation done 47 days previously.

Blood transfusion was started soon after admission, and, after an adequate quantity had been cross matched (6 pt.), the patient was taken to surgery at 2:20 a. m., April 11, 1950. The abdomen was opened through a generous right paramedian incision and was found to contain a huge quantity of blood, bright and dark, which replaced itself rapidly on evacuation. The blood was found to be entering the abdomen through a hole 2 cm. in diameter in the posterior parietal peritoneum overlying the right nephrectomy site (Figure). There was a tense hematoma behind the duodenum and pancreas and liver, and the blood entered the abdomen not with force or pulsation but with alarming persistence. There seemed, at the moment, to be no good solution to the problem, because to evacuate the hematoma would release the tamponade and spell almost certain disaster.

It occurred to us at this moment that the propulsive, purposeful flow in the vena cava in the prone position is from below upward, whereas the bleeding from above is the result of pressure in the right auricle projected two to three feet from the heart and modified by the negative intrathoracic pressure. It is not directly the result of systole and diastole. With this in mind, the mesentery overlying the inferior vena cava was opened and the left renal vein was identified, to make certain that the ligature would be placed below it. A heavy silk ligature was then passed under the vessel and, when it was lifted, the bleeding stopped. This was repeated several times to be certain of cause and effect. The vena cava was then ligated with three separate silk ligatures; the hematoma was opened and evacuated; a light dry gauze pack was applied and brought out through a stab wound, and the abdomen was closed in layers.

The patient made an uneventful recovery. The pack was removed on the fifth postoperative day without incident. The patient was up and about on the eighth day and left the hospital with a urea nitrogen level of 11.2 mg. per 100 cc. and a red blood cell count of 3,700,000, with 11.0 gm. of hemoglobin, on May 3, 1950, twenty-three days after surgery. Within 60 days the leg edema incident to ligation was minimal, and this woman, now 73 years of age, continues to lead an active life. Urea nitrogen one year later was 12.0 mg. per 100 cc., and red blood cell count was 4,300,000, with 12.7 gm. of hemoglobin.

## SUMMARY AND CONCLUSIONS

Ligation of the inferior vena cava is presented as a means of controlling late postnephrectomy hemorrhage. We believe this to be the first instance of ligation for this indication. One wonders if this might not be of help to the urologist in the management of postnephrectomy venous hemorrhage at any stage or in the control of hemorrhage from the renal veins or vena cava traumatized from any cause. The slow steady bleeding from the proximal end of the vena cava can easily be controlled by light pressure.

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## EDITORIAL: LIGATION OF THE VENA CAVA

The earlier examples of ligation of the inferior vena cava in man were the result of accidental injury, usually during nephrectomy. O. G. Pfaff credits Battim with the first successful ligation, in 1893. Ligations which terminated fatally had been reported by Kocher, by Billroth, and by Kiester. More than a hundred years earlier, Haller (1756) demonstrated the collateral circulation in a case of thrombosis of the inferior vena cava. In more recent years this blood vessel has been ligated more frequently in the treatment of thrombophlebitis or severe pelvic infection. Injuries to the vena cava reported during the past twenty-five years in most cases have been treated successfully by suture or by clamping the injured area with one or more curved hemostats which were left in place five to seven days.

In the preceding article an interesting case of delayed postnephrectomy hemorrhage controlled by ligation of the inferior vena cava is reported. Although the results in this case give further proof that the inferior vena cava may be safely ligated, it is a drastic procedure, to be used only when suture or forcipressure cannot be safely instituted. Hyman and Leiter credited Wakefield and Mayo with collecting nineteen cases of ligation of the vena cava. Five of these patients died, and in the remaining fourteen eight had edema of the legs, in five of whom it persisted. This indicates that the collateral venous channels are more adequate in some individuals than in others.

Walters and Priestly found in their experimental work on animals that the vena cava could be opened, and closed with either suture or clamp without evidence of extensive thromboses. In all animals there was a satisfactory lumen after four weeks, and there were no pulmonary infarcts or emboli. They reported four successful cases of suture of the vena cava during nephrectomy.

Pfaff, who is referred to in the report by Drs. Bolman and Lloyd, collected thirty-seven cases from the literature. In nineteen of these total ligation was done, with six deaths. Lateral ligation was done in seven patients with two deaths, and one patient whose wound was packed died. Five patients in whom the wound in the vena cava was sutured and six in whom the injury was treated by forcipressure recovered. Regardless of the fact that most of the fatalities in this series occurred too early for the ligation to have been a factor, it is difficult to reconcile Pfaff's conclusion that ligation was a safer procedure. The pressure in the vena cava is low, and bleeding can be readily controlled by digital pressure or the temporary application of Serefine or rubber-covered clamps while the laceration

**Received** for publication Nov. 22, 1955.

is sutured or the margins approximated by curved forceps. With modern methods of sustaining the patient, time is rarely a serious problem. Clamps can be applied as quickly as a ligature. Their only disadvantage is discomfort to the patient during the early days of convalescence. Secondary hemorrhage has not been reported at the time of removal.

In our opinion postnephrectomy bleeding or hemorrhage occurring during nephrectomy can usually be controlled by ligature or suture. Ligation of the vena cava should be reserved for those cases in which, because of poor exposure, lack of adequate assistance, extensive laceration, or extensive bleeding, the time saved by ligation is important in saving the patient's life.

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# Diseases of the Gall Bladder and Bile Ducts

**Surgical Aspects**

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It is not my intention in the time at my disposal to review the diagnosis and treatment of the known and well-understood lesions of the gall bladder and bile ducts. On the contrary, it is my desire to call your attention to some of the controversial points in the diagnosis and therapy of lesions in this region.

There is probably no more fascinating field of medicine than the biliary tract and the various aspects of lesions which may affect it. The reason for this is the fact that a lesion in the biliary tract is hardly ever entirely localized, since portions of the tract are present within the liver and the lower portion of the common bile duct is surrounded by the head of the pancreas in two of three cases. It is apparent that inflammation in the wall of the gall bladder or the bile ducts is likely to extend through them to the structures in which they begin, course, and end. The disturbances of the biliary tract, broadly speaking, are those due to infection and obstruction; each can produce the other. In many instances they occur together.

Submitted for publication Oct. 5, 1955.

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Read as part of a Panel Discussion on Diseases of the Upper Abdomen in the General Scientific Meetings at the 104th Annual Meeting of the American Medical Association, Atlantic City, June 6, 1955.

## CHOLECYSTECTOMY

There is little need to emphasize the fact that the patient having severe attacks of biliary colic should have the diseased gall bladder removed. On the other hand, some physicians and surgeons, I am afraid, still believe that when gallstones are not producing symptoms there is no particular need or urgency in having the gall bladder removed.

This viewpoint is difficult for me to understand, for stones in the gall bladder will most certainly at some time produce obstruction of the cystic duct, or they may pass into the common duct and produce varying degrees of infection within the biliary passages and obstruction. The risk on operation is thus increased, but, worse than that, the infection within the intrahepatic bile radicles and in the pancreas will increase with time and may continue to cause troublesome symptoms after the delayed removal of the diseased gall bladder or the common-duct stones. So, for many years I have advised cholecystectomy in patients who have gallstones, even though they have had no colics and only minor symptoms of dyspepsia. The risk of cholecystectomy should be less than 1%. If, on the other hand, obstruction of the cystic duct occurs and produces a swollen, reddened, edematous gall bladder or if a stone obstructs the common duct and produces jaundice, a surgical procedure directed to the elimination of these disturbing and painful lesions carries a risk three and a half times greater.

*Presence of Cardiovascular Disease.*—And what of the patient who urgently needs cholecystectomy because of recurring and severely painful attacks of biliary colic and who has been handicapped as a result of

cardiovascular disease, particularly coronary thrombosis? Keys, Dry, Walters, and Gage [1] recently studied the records of 100 patients who were operated upon at the Mayo Clinic because of gall-bladder disease and who had associated coronary heart disease. They hoped to determine the risk of cholecystectomy for such patients and the influence of cholecystectomy on the subsequent course of the patient, particularly with regard to the heart. There were three postoperative deaths in the group. One was the result of acute pancreatitis and occurred on the fourth day after operation. The second was due to a pancreatic necrosis and occurred the 21st day. This second patient had survived pulmonary embolism on the seventh postoperative day. Both of these patients had acute infection in the gall bladder associated with obstruction. The postponement of the operation in these cases had led to the extension of the infection into the pancreas. The third death occurred on the 17th postoperative day. It was due to cardiac failure and was associated with a cerebral vascular accident. This risk of 3% is a little lower than that reported by Brumm and Willius [2] in 1939 in their study of surgical risk for patients with coronary diseases. Their patients had had coronary thrombosis. Major surgical procedures were carried out on them with a risk of 4.5%.

As far as a beneficial effect of the cholecystectomy on the coronary heart disease is concerned, Keys, Dry, Walters, and Gage could find no direct evidence that cholecystectomy improved the function of the heart. One could speculate that the cholecystectomy did prevent development of any serious complications in the biliary tract which might have terminated the patients' lives. Study of these cases by actuarial methods showed a seven-year survival rate of 70.6% after cholecystectomy, in contrast to 83.9% for the normal population of the same age and sex.

### STRICTURE OF THE COMMON DUCT AND BILIARY FISTULA

Since the war, my colleagues and I [3] have seen and operated on a distressing number of patients with strictures of the common bile duct with and without external biliary fistulas. The number of these seems to be increasing; last year we operated on 76 such patients. Almost without exception the stricture had followed an operative procedure on the gall bladder or bile ducts, and this complication should not have occurred.

The operation of cholecystectomy should be done only by surgeons who are absolutely certain of their ability to dissect out and demonstrate the cystic duct, the cystic artery, the common duct, and the common hepatic duct. Anomalies of these structures are common, and it is difficult, especially in the presence of an acute inflammation involving the gall bladder and the bile ducts with which they may be so intimately associated, to separate one from another. In such cases, it is always better, in my opinion, for the surgeon with limited experience to be content with removal of the gallstones and drainage of the gall bladder, allowing the inflammation to subside and doing the cholecystectomy at a later time.

Before leaving the discussion of strictures, I should like to comment on the repair of strictures and on the value of injecting radiopaque substances into the biliary fistulous tract before operation.

Since 1924, when I first became interested in operating for strictures of the common bile duct, I have performed 385 surgical procedures for this unfortunate and serious type of lesion. Generally speaking, the hospital mortality rate has averaged about 4%. The type of operation which in my hands has given the best results has been that in which the ends of the bile ducts were anastomosed or the end of the common duct above the stricture was antomosed to the duodenum and a prosthetic device of some sort, either an indwelling catheter or portion of it or a T-tube, was left in place for 9 to 12 months to prevent contracture of the anastomosis. My results have been excellent in approximately 70% to 75% of cases, with no further evidence of biliary obstruction. Of the patients, 15% have had mild symptoms of biliary obstruction, and 10% have had symptoms so severe

that they required reoperation. When there is a great amount of intrahepatic infection with intrahepatic biliary stones or when the patient has been deeply jaundiced or both and there is considerable disturbance in the liver, I have preferred to use the T-tube for drainage, since I could be assured of complete relief of biliary obstruction and could have the opportunity of keeping the intrahepatic ducts free of recurring stone formation by frequent irrigation through the T-tube.

In the presence of a biliary fistula, a small catheter introduced into the fistula keeps the abdominal wall free of bile-stained secretion. Given through this tube, a radiopaque substance, such as iodopyracet (Diodrast) or sodium acetrizoate (Urokon), will usually show in a roentgenogram where the obstruction is. Often such a roentgenogram demonstrates lack of continuity of the duct as a result of disruption, and infrequently it shows the shadow of a stone in the common duct or a pancreatic obstruction if either is the cause of the stricture. The preferable time to repair a common bile duct that has lost its continuity as a result of operation is at the time of operation. Should this unfortunate accident occur, an immediate repair by suturing the ends of the ducts together and using a T-tube to serve as a temporary splint, or a catheter passed through the lower end of the duct into the duodenum, is an effective method of assisting in healing without contracture of the anastomosis.

### OTHER TYPES OF OBSTRUCTION OF THE COMMON DUCT

Obstructions of the common duct are due, for the most part, to stones, stricture, malignant lesions of the ampulla of Vater or the head of the pancreas, inflammatory lesions of the head of the pancreas or in the intrahepatic tissues surrounding the biliary radicles, and inflammation, stenosis, or spasm of the sphincter of Oddi. The stone in the common duct which produces sufficient obstruction to cause, in addition to pain, intermittent fever or jaundice is not difficult to diagnose. On the other hand, it must not be forgotten that a third of the patients with stones in the common duct have neither chills nor intermittent fever, and two-thirds may not have had jaundice.*

The new radiopaque crystalline substance, Cholografin, has been very helpful in showing the presence of stones in the common duct that are larger than 1 cm. in diameter. My colleagues in the Section of Roentgenology at the Mayo Clinic have made Cholografin studies on more than 250 patients. At this time, they hold about the same opinion as that expressed by the French surgeons last year, namely, that there is a 16% error in the interpretation of the Cholografin images. This error is due to the faintness with which the duct is outlined by the Cholografin in some cases and to the occasional small accumulations of gas in the gastrointestinal tract which will cause misleading interpretations. Studies with Cholografin have definite value, however, especially when positive, and interpretation of the shadows is of real value in some cases as an aid to a thorough clinical evaluation.

For example, in November, 1951, I performed an end-to-end anastomosis of the common bile duct in a patient who had a stricture. She has had an excellent result from the operation; there have been no evidences of biliary obstruction, but recently she has had some discomfort in the right upper quadrant of the abdomen. An x-ray examination of the abdomen showed air in the biliary system. The Cholografin study revealed the outline of the common bile duct above and below the point of anastomosis to be of normal size, and the duct seemed to be emptying satisfactorily. This verified the clinical impression that her abdominal discomfort was not due to biliary obstruction at the site of the anastomosis.

Roentgenologists at the Mayo Clinic have indicated also that use of the tomograph has

* Trueman, K., cited by Walters, Snell, and Clagett.[4]

helped improve the accuracy of their diagnoses. The patients with the typical syndrome of stones in the common duct who actually do not have stones in the common duct usually have one of the other lesions which I have listed.

Since the preoperative diagnosis of stone in the common duct is not always possible, it is obligatory whenever cholecystectomy is performed for the surgeon thoroughly to search the common and hepatic ducts for stones or other obstruction if the common duct is enlarged. He should open the common duct to do this. He should pass a 4 mm. and if advisable a 6 or 8 mm. probe or scoop through the lower end of the duct into the duodenum without meeting an obstruction and palpate the duct over the probe for stones. The head and body of the pancreas should always be palpated. If a stone is found, the procedure is clear enough.

If no lesion or resistance of any kind is found, the obstruction may be intrahepatic and not infrequently it is of the type described by Watson, Hoffbauer, and Howard [5] as pericholangitic. The diagnosis can be confirmed by removal and microscopic examination of a small bit of liver.

I have seen and operated on several patients who had pericholangitic obstruction. The clinical picture, as well as the laboratory data, indicated biliary obstruction but did not localize it to the liver. Indeed, I have seen patients with this lesion, accompanied by an increase of fibrosis within the liver, whose attacks of pain associated with the jaundice simulated those of extrahepatic biliary obstruction. Interestingly, many of these patients with pericholangitic obstruction have improved after prolonged T-tube drainage of the common duct. Itching frequently is a serious and disabling symptom. Strangely enough, after drainage of the common duct, itching tends to subside and sometimes disappears almost completely.

When the exploring scoop in the common duct does not readily pass through the ampulla, the surgeon must ascertain by opening the duodenum whether he is dealing with a papillary carcinoma of the lower part of the common bile duct or early carcinoma of the pancreas. If a lesion is present at the ampulla and the condition of the patient permits, a radical resection of the area and transplantation and reimplantation of the divided structures, as advocated by Whipple [6] and Parsons,[7] Brunschwig,[8] Waugh,[9] and others, should be carried out.

When there is uncertainty whether an enlargement in the head of the pancreas is benign or malignant, if it is stony hard and small and confined to the head of the pancreas, we remove the lesion after first trying to obtain an accurate specimen of the entire thickness of the tumor to determine the presence or absence of neoplasm. The results in the management of such cases have recently been surveyed at the Mayo Clinic by Waugh and his assistant Giberson.† They found that the five-year or longer survival rate when radical resections were done for ampullary lesions was 38%. In the cases of carcinoma in the head of the pancreas in which an attempt had been made to remove too extensive and too fixed malignant tumors, the results were but little better than they were when the conservative operation of biliary-intestinal short circuit had been carried out. In recent years, however, when the radical resection has been confined to small, movable, localized malignant tumors in the head of the pancreas, the results have proved to be much better and 15.8% of these patients are living five years or longer after operation.

The most puzzling cases are those in which attacks of what seems to be biliary colic still occur after cholecystectomy and no lesion or stones are found on opening the common duct. If intrahepatic pericholangitis can be ruled out, the questions to be decided are whether fibrosis of the sphincter of Oddi is present and is producing intermittent inflammation and biliary obstruction, whether painful seizures can be the result of spasm of the sphincter of Oddi or inflammation of

†Giberson, R. G., and Waugh, J. M.: Unpublished data.

it, and also whether, as a result, the intrabiliary pressure is increased sufficiently to allow bile to enter the pancreatic duct and produce pancreatitis. I elaborated more fully on this important subject in my paper [10] before the Section on Surgery of the American Medical Association, in June, 1955. For this article I shall merely say that there seems to be definite evidence in certain cases that, as a result of inflammatory lesions of the biliary tract, stenosis and inflammation do occur in and around the sphincter of Oddi and the papilla of Vater. In these cases, an increased pressure occurs within the common duct and produces attacks of pain.

Such attacks were reproduced clinically many years ago by McGowan, Butsch, and myself.[11] When the increase in intraductal pressure was the result of spasm of the sphincter of Oddi, we showed that glyceryl trinitrate given sublingually would reduce the intrabiliary pressure and relieve the pain. Morphine and codeine, on the contrary, would produce pain because they increased intraductal pressure, which causes the pain.

In many of these cases division of the sphincter of Oddi will give the patient relief of pain. In other cases it will not. The reason probably is that constriction by the inflamed portion of the pancreas through which the common bile duct passes continues to produce biliary stasis. I referred to this in 1953 in my discussion on a paper by Cattell and Colcock [12] on spasm, inflammation, and fibrosis of the sphincter of Oddi. I had demonstrated the presence of this narrowing of the pancreatic portion of the common duct in two cases in which dilatation of the common duct and biliary stasis continued to occur after sphincterotomy.

More recently, with the aid of a short T-tube to study the physiology of the common bile duct after sphincterotomy, my colleagues and I were able to reproduce the attacks of biliary colic by injecting a radiopaque thorium dioxide preparation called Umbrathor into the common duct via the T-tube. A cholangiogram showed spasm of the common duct above the site of the sphincterotomy, with reflux of the injected material into the pancreatic duct. Glyceryl trinitrate administered sublingually temporarily relieved this attack of pain in the first case we studied. The pain recurred after a short time and was partially relieved by meperidine, although spasm of the duct continued. Two hours later we were able to aspirate duodenal contents from the common duct through the short T-tube, and a study revealed no sustained intraductal pressure. A few days later we repeated the study and found that with the attack of pain intraductal pressure was increased to more than 300 mm. of water. At that time the spasm of the lower end of the common duct and the reflux of radiopaque material into the pancreatic duct were noted in the roentgenogram. The patient's pain was epigastric and severe and similar to the pain she had had before operation. Needles were inserted into the left and right splanchnic regions, and procaine hydrochloride was injected to block the left splanchnic nerve. Within three seconds the pain was relieved. X-ray examination at this time still showed spasm of the lower end of the duct, with retention of medium in the common duct and in the pancreatic duct. The intraductal pressure was still high and static at about 250 mm. of water. Shortly afterward, a second injection of the Umbrathor was given to increase the intraductal pressure. The epigastric pain did not return, but the patient had a feeling of fulness to the right of the incision. The right splanchnic nerve was not blocked with procaine at this time.

It is possible that the feeling of fulness and the pain were visceral in origin and were due either to distention of the duct or to distention or contracture of the first part of the duodenum. This study seems to show that spasm of the duct will cause attacks of pain simulating those caused by spasm of the sphincter of Oddi, and, even though sphincterotomy has been done, ductal spasm may cause reflux into the pancreatic duct. This spasm and the resultant reflux may be

responsible for failure of sphincterotomy to relieve attacks of pain in some cases.

The relation of reflux of bile into the pancreatic duct as a result of increase in intraductal pressure is somewhat confused by the fact that Kleitsch [13] in a recent study of the anatomy of the main pancreatic ducts demonstrated that in 10% of the cases the main pancreatic duct empties into the duodenum through a separate ampulla caudad to the ampulla of Vater and in an additional 60% of cases a large accessory branch drains the main pancreatic duct, which also has a separate opening in the duodenum. This makes a total of 70% of the cases in which an increase in intraductal biliary pressure with reflux into the pancreatic duct would not increase intraductal pancreatic pressure. Moreover, recently, Parry, Hallenbeck, and Grindlay,[14] using a new method of measuring intraductal pressure without obstructing the duct in dogs, have shown that pressures within the pancreatic duct exceed those within the common bile duct.

Further confusing the pressure mechanisms in the Vaterian ampulla is the fact that in some of the cases in which I have done sphincterotomy and have searched for the opening in the pancreatic duct a flap of mucous membrane has been lying over the opening of the duct. With an intact sphincter of Oddi, an increase in intrabiliary-duct pressure would tend to close the ampullary opening into the pancreatic duct by pressing the flap over it.

The important fact remains, however, that a patient having attacks of biliary colic subsequent to sphincterotomy, in which spasm of the common duct occurs with reflux of the radiopaque substance into the pancreatic duct, was relieved of pain as a result of injection of procaine into the left splanchnic nerve. In other cases when the pain was relieved in this way, an injection of a small amount of alcohol sufficiently injured the nerve fibers to give continued relief of pain for periods of four or five months. This method of giving prolonged relief of pain was used on two separate occasions in one of my patients who had not had sphincterotomy but had had a previous prolonged T-tube drainage of the common bile duct which had also prevented the painful attacks.

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# Diseases of the Liver

## Medical Aspects

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Medical aspects of liver disease include the great majority of diagnostic and therapeutic procedures possible at this time. While biopsy at laparotomy is still a frequent, very useful method for diagnosis and despite recent bold attacks upon malignant disease of the liver with whole-lobe resections, most diseases of the liver are managed by medical men and not by surgeons. The one outstanding exception is biliary cirrhosis due to extrahepatic obstruction.

Our most frequent tasks include diagnosis and management of cirrhosis, the differential diagnosis of jaundice, and the problem of hepatitis. There is a growing awareness of our responsibility for prophylaxis against certain types of liver disease, notably cirrhosis and serum hepatitis. To a smaller extent but still worthy of long-range planning, one might forsee better control of the hygienic and other factors which may eventually be identified with infectious hepatitis. The evidence that gamma globulin can confer reasonably dependable temporary passive immunity to hepatitis is a big step in this direction and is already receiving wide utilization in epidemic outbreaks.

Received for publication Oct. 10, 1955.

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Read as part of a Panel Discussion on Diseases of the Upper Abdomen in the General Scientific Meetings at the 104th Annual Meeting of the American Medical Association, Atlantic City, June 6, 1955.

The incidence of cirrhosis varies widely, with racial and geographical differences that are not easy to understand. The nutritional factors which so often depend upon natural resources and their development and availability to a population probably represent the only explanatory common denominator for these differences. In America cirrhosis should be rare, since few go hungry, but we all know this is far from true. If cirrhosis is essentially a deficiency disease, it is certainly our most frequent and important one. The role of alcohol is still unknown, but many factors cause us to feel that it is not merely coincidental in cirrhosis as we see it in our daily practice. Few, if any, patients can make real progress or maintain improvement in their liver functional state if they do not abstain from alcohol entirely.

With few exceptions, cirrhosis is not difficult to diagnose. Rarely must one resort to biopsy for diagnosis, but it is often useful in ruling out additional or superimposed disease in a clinically obvious cirrhotic patient. The history of increasing fatigue and (in the absence of ascites) weight loss in a patient who uses more than the average amount of alcohol should make one at least as suspicious of cirrhosis as of tuberculosis or malignant disease. When arteriolar "spiders," red palms, firmness and palpable enlargement of the liver, and faint persistent icterus are noted, other disease may coexist but cirrhosis is almost certainly present. Mild degrees of cirrhosis show none of these signs and generally cause no symptoms at all. If they are to be detected in the course of routine physical surveys, it will be possible only because a history of hepatitis or significant alcoholism led to the decision to perform a

biopsy. Jaundice or ascites signifies extensive hepatic-cell damage with or without portal hypertension and, together with the signs described, leaves little doubt as to diagnosis. Even in these "decompensated" cirrhotics, treatment may lead to sustained improvement if alcohol is discontinued. Hemorrhages from dilated veins of the portal system or collaterals signify a much poorer prognosis. Even in this serious complication, however, if enough functioning hepatic cells survive, surgical anastomoses may successfully prevent further hemorrhage, and medical treatment may produce good long-term results. The ultimate in decompensation, hepatic coma, is only very rarely followed by recovery and long survival thereafter.

Treatment of mild to moderate (or "compensated") cirrhosis consists of only three things: (1) absolute abstinence from alcohol, (2) increased physical rest, and (3) intake of a full well-balanced dietary (or its equivalent in special supplements or substitutes as circumstances may require). Infections, fatigue, and trauma, particularly if associated with loss of blood, must be considered much more seriously than usual in cirrhotics, since any of these may lead to "decompensation" through hepatic-cell oxygen and nutritional deprivation and necrosis. Herein lies the all-important factor of success or failure in the medical management of the average cirrhotic. Elective surgery should always be avoided. Ambitious work projects should be discouraged. A real effort should be made to teach the patient how to live within his cirrhosis-imposed limitations. For the patient this is often difficult, for his safe course is not as well marked as that of the patient with angina or hypertension, and he must learn to recognize fatigue at his most important warning. When jaundice appears, bed rest is undoubtedly indicated but often difficult to continue if recovery is prolonged, as is so often the case. Physical activity when jaundice signifies advancing cirrhosis is always harmful and may lead to further and rapid decline.

Ascites is not well understood in all its complex aspects but strongly suggests that significant portal hypertension exists along with hepatic-cell failure in the important area of protein synthesis. Although most unpleasant to the patient and demanding of his complete cooperation, sodium restriction fortunately leads to improvement in most patients, often with complete disappearance of ascites. No other single factor is so important. Mercurial diuretics offer valuable assistance, and paracentesis should be avoided as long as possible. In general, serious impairment of ability to eat or breathe constitutes the regrettable but inescapable indication for paracentesis. To see the hard-won protein irretrievably lost by this procedure hurts us in the same fashion as the sight of the nephrotic's protein rich urine. Surgery for the relief of ascites has been disappointing, but many who feel that mechanical factors are paramount in causation urge portacaval anastomoses whenever feasible. Perhaps more will be done in the future if nonoperative methods of determining portal pressures become safer and more dependable and if a larger number of cases can be collected to bear out the contention that portal hypertension is a major cause and not merely a coexistent problem in the average patient with ascites. This remains to be proved, and there is much evidence against the idea.

Use of corticotropin, steroids, and testosterone in the adjunct treatment of more serious degrees of cirrhosis has many proponents. Generally, while transient benefits may appear to result, long-term effects are unchanged. In precoma and coma corticotropin (ACTH) particularly can sometimes achieve dramatic temporary improvement. If ammonia-containing foods are used sparingly and if absorption of ammonia is prevented with sulfonamides or antibiotics which reduce intestinal bacteria population, some prolongation of the essentially temporary benefit may be brought about. In general, it is true that early or moderately advanced cirrhosis may be managed quite successfully if patient-cooperation can be obtained. Decompensated cirrhosis, with jaundice, ascites, varix bleeding, and various degrees of central nervous

system damage, may be influenced to some degree by available treatment measures, but long-term results are still disappointing.

The history of the jaundiced patient must be emphasized as the most important single differential diagnostic aid. Duration, fluctuation, pruritus, pain, chills, fever, anorexia, weight loss, antecedent trauma, exposure to epidemics, or percutaneous injections all bear careful evaluation. Examination may reveal a palpable mass, the stigmata of cirrhosis, and a liver of variable size, shape, and tenderness. Probably 90% or more will prove to be correctly diagnosed by history and examination alone.

The liver profile need not be elaborate or costly. Bilirubinemia itself proves incapacity of secretion and excretion. The suspicion that a clay-colored stool means no bile flow can be confirmed easily by the qualitative Wallace and Diamond test for urine urobilinogen, which by serial dilution of urine becomes a fairly accurate quantitative test as well. It is worth remembering that oral antibiotics may (rarely) alter reducing bacteria in the intestine sufficiently to influence urobilinogen formation, leading to an erroneous impression that no bilirubin is entering the bowel.

Within the first three or four weeks of jaundice, tests which indicate failure to synthesize normal globulins and phospholipids are more likely to be strongly positive in hepatitis than in extrahepatic obstruction. Thus, the various turbidity and flocculation tests offer valuable differential service. They may well be positive in cirrhosis also, which makes it difficult to be sure of hepatitis superimposed in these patients. Alkaline phosphatase is excreted in bile, as is cholesterol. Increases in serum concentrations of these may occur in hepatitis during the period of greatest hepatic enlargement, but persistent elevations or higher concentrations point toward extrahepatic causes. Serum iron is rarely significantly high, except in hepatitis. This test may ultimately be of increasing value.

Biopsy offers the best nonoperative means of differential diagnosis in cases of serious doubt. After prolonged extrahepatic obstruction, bile ducts within the liver may reach large size and contain bile under greatly increased pressure. The misfortune of striking one of these with the biopsy needle is frequently attended with serious morbidity and occasionally mortality. This complication may be lessened by proceeding with the biopsy before many weeks of jaundice have passed. Most hepatitis patients reach their peak of bilirubinemia in three weeks and begin to decline thereafter. Uncertainty after four weeks of jaundice probably should lead to biopsy in most cases. In spite of all these measures we still operate upon patients with hepatic jaundice and postpone surgery overlong in those with extrahepatic obstruction. Our only comfort is the fact that we do so less often than formerly; particularly in the days before the biochemical tests and especially in the days before needle-aspiration biopsy.

Hepatitis-producing viruses A and B still appear to be as far as ever from any sort of control. When sporadic cases of infectious hepatitis appear, it might be wise to extend the investigation to close relatives or associates with a flocculation test or two, since may small epidemics of mild hepatitis undoubtedly go undiscovered. Protection of the very young or old, the debilitated or those pregnant can be afforded with gamma globulin. Probably we should use it oftener. Diagnosis is strongly suggested by the week or two of malaise with headache and digestive "upset" which usually precedes the appearance of jaundice. Too often this history is lacking, and occasionally it means infectious mononucleosis, but, if sought, it can often be brought out from an otherwise forgetful or phlegmatic patient.

The tender liver, usually somewhat enlarged, is most helpful. It is not encountered early in obstructive jaundice and is seldom present without ample additional signs in cirrhosis. In rare cases of hepatitis, pruritus may occur and even ascites of detectable degree. If these are seen in one who has

lost weight owing to several weeks of anorexia or vomiting, the puzzle is a difficult one. Unless the clinical course points clearly to subsidence of hepatic reaction with unmistakable improvement in condition and laboratory picture as well, biopsy should not be postponed too long. In the usual case of hepatitis biopsy is not required for diagnosis. Even before impairment of bilirubin excretion becomes obvious, protein synthesis is impaired and fat metabolism altered. Flocculation tests usually become rather strongly positive within the first two weeks.

Biphasic curves of urobilinogenuria occur with increases due to failure of hepatic-cell excretion and a peak-of-disease reduction or even temporary absence due to cessation of bile flow caused by intrahepatic compression. During convalescence, occasional sharp increases of urobilinogenuria may be observed if they are looked for. The prolonged absence of urobilinogen from urine, which is sometimes seen, often means canalicular thrombi of dried bile. These may be seen with considerable perilobular round-cell infiltration of a type not dissimilar to that in early biliary or obstructive cirrhosis. Their real significance in hepatitis is not clear, and their earlier appearance in some of the drug-induced jaundices (without cellular reaction) may be more than coincidence. Follow-up biopsy on these cases after recovery does not suggest true biliary cirrhosis, and those cases in which exploratory operation was performed have shown no extrahepatic obstruction. For the time being, many continue to label these cases "cholangiolitic hepatitis," as suggested by Watson and Hoffbauer. Fortunately they occur rarely.

The majority of hepatitis patients are only mildly or moderately ill. The few who are seriously ill and who are likely to have a delayed convalescence or even the permanent sequelae of posthepatitis cirrhosis cannot be distinguished early in the course of the disease. The majority, therefore, require a minimum of supportive treatment and may well remain ambulatory throughout. The few, however, seem to obtain valuable protection from rather intensive supportive measures, including complete bed rest. It is difficult to find any safe alternative to the prescription of bed rest for all who are seen early in the course of their disease.

When the clinical course is rapidly favorable and when bilirubinemia clearly begins to decline, most patients may resume normal physical activity on trial. If fatigue is noticeable or if aching and tenderness of the liver are unmistakable, a prompt retreat to bed is in order. Otherwise, normal activity, including nonstrenuous work, does not appear to affect convalescence adversely or to interfere with complete recovery. A full well-balanced diet must be taken throughout the illness. Often this is difficult during the early weeks, and needed ingredients must sometimes be provided by intragastric catheter or by parenteral routes temporarily.

There are no specific antiviral agents for hepatitis A or B. While corticotropin and cortisone almost always reduce bilirubinemia promptly and may be responsible for better appetite and a sense of well-being, they do not seem to alter the course of the disease permanently and quite a few relapses occur when treatment with these hormones is stopped. Smaller doses have been given with reported good results in shortening over-all duration of illness, but relapses must still be looked for. Further experience may alter prevailing opinion, but most observers feel hormones should not be a part of the treatment program.

Except for a history of exposure to blood or blood derivatives and an appropriate interval for incubation, there is no practical way to distinguish serum hepatitis from its more frequently encountered twin. This causes many medicolegal squabbles and adds gray hairs to the Workmen's Compensation Board judge. When doubtful cases are heard, a clear history of possible exposure is usually accepted as proof of the diagnosis, although everyone knows this is not always true. Generally speaking, morbidity is greater and sequelae are more frequent in hepatitis B. Treatment is of course the same. Most of

these patients should be observed with greater care and followed over a longer period of time because of the somewhat more serious nature of the disease and also because of the frequent suits for damages or compensation which follow.

Intensive efforts to screen donors of blood for carriers of B virus have met with only fair success. Proof that carriers exist with no history of jaundice whatever has recently been provided and is, of course, most discouraging. Efforts to destroy or inactivate B virus in blood or serum have not been successful. There is evidence that some diminution in virulence occurs when virus-containing plasma is stored at room temperature for long periods, but this is hard to prove conclusively. Fortunately, fibrinogen and gamma globulin have thus far not been incriminated as carriers of B virus. At this time, the sole method of prophylaxis is the withholding of blood and blood products (with the exceptions noted), unless the emergency really justifies the risk.

# Diseases of the Pancreas

## Surgical Aspects

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One cannot cover an all-inclusive viewing of the possibilities of surgery for pancreatic conditions in a presentation of this nature. No doubt many of the points will be brought out in the panel discussion. This short discourse will attempt to name the important well-known facts about pancreatic surgical conditions and to mention those aspects which are presently being tossed about in a spirit of controversy.

One of the least known of the many advances in surgery during the past two decades is the progress in surgery of the pancreas. Twenty-five years ago very little was accomplished for the benefit of the patient who had some of the conditions mentioned below. The triumphs are muted only by the rarity of the conditions and the more glamorous management in the important cardiovascular surgical improvements. Nevertheless, these patients represent a sizable group of invalids who cannot now properly continue to suffer the neglect or disinterest of the profession. Formerly, it was not necessary to exercise concern and expend energy on these conditions because each physician realized that other physicians as well could do nothing for the benefit of these patients. Today, so much is known and so much can be done that the physician who hesitates to trouble himself with concern about patients with pancreatic conditions can really be accused of either extreme ignorance or neglect.

The conditions of surgical interest in the pancreas are similar to those in other organs, i. e., the congenital, traumatic, infectious, and neoplastic disorders. It is hoped that this discourse may be helpful in clarifying the working principles of pancreatic surgical conditions.

### CONGENITAL DISORDERS OF THE PANCREAS

True pancreatic cysts are epithelial-lined and are rarely seen. They are undoubtedly the result of developmental malformation and ostensibly are retention cysts. The treatment is excision if technically feasible, internal drainage by anastomosis of the cyst to the stomach or intestine, or, if neither of these can be accomplished, marsupialization, which is actually external drainage and the procedure yielding the poorest results. As experience is gained, the cystogastrostomy or cystoenterostomy (Roux-Y type) is being universally accepted. No undesirable aftereffects have been reported.

*Annular Pancreas.*—In this condition the duodenum is encircled by a ring-like tongue of pancreatic tissue. This produces duodenal obstruction and may not exert its full obstructive power in childhood; the symptoms often appear in adults. The duodenal obstruction permits an easy diagnosis, but the true annular pancreas nature is most usually discovered at the operation. The operative procedure is simply duodenojejunostomy, which yields correct results without danger. Surgical interruption of the ring or attempt at removal may well induce pancreatic fistula or necrosis, which may be fatal complications.

*Aberrant Pancreas.*—These misplaced developmental pieces of pancreas can be seen

Received for publication Oct. 10, 1955.

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Read as part of a Panel Discussion on Diseases of the Upper Abdomen in the General Scientific Meetings at the 104th Annual Meeting of the American Medical Association, Atlantic City, June 6, 1955.

in the duodenum, stomach, small bowel diverticula, transverse mesocolon, jejunal mesentery, or rarely in some peritoneal or adipose structures adjacent to the pancreas. They can undergo any pathologic change experienced by the parent organ, even infection, although tumors are the commonest pathologic change. Symptoms are produced according to location and usually by pressure upon a viscus or organ. Surgical removal of aberrant pancreatic tumors is indicated, and usually the operation is not so difficult or dangerous as tumor removal from the main organ. It is believed that many small aberrant pancreatic bits of tissue remain inactive during life, produce no symptoms, and are often unrecognized during postmortem examination. Clinical confirmation of the last statement can occasionally be had when total pancreatectomy can be done and little or no substitutive therapy is necessary after the operation.

### INJURIES TO THE PANCREAS

One sees two types of trauma: the closed injury, when the pancreas ruptures because of compression or blast phenomenon, and the direct injury due to missiles, stabbing instruments, or accident during surgical operations for nonpancreatic upper abdominal conditions. The surgeon's conduct in these instances is rather clearly defined.

At laparotomy for nonsurgical trauma it may be difficult to know that the pancreas has been injured. Any retroperitoneal or peripancreatic hematoma must be considered to be a pancreatic injury, unless inspection of the pancreas is so satisfactory that the operator can be certain that the pancreas is unhurt. This is important. Obviously, when a tear or perforation is observed, bleeding must be controlled and an attempt at repair is usually feasible but must be done with great care. The same goes for surgical trauma. But the matter of most importance, when the bleeding has been controlled, is that of drainage. It is absolutely necessary if safety is to be secured because fatal pancreatitis may promptly and disastrously ensue. It is better to have a pancreatic fistula which, although undesirable, exists in a living patient but which can often be controlled than to have an accumulation of pancreatic juice in the peritoneal cavity too great in amount for human survival. Thus, the important procedure, even if injury is only suspected, is drainage. It will be a life-saving drainage more frequently than one realizes.

### INFECTIOUS CONDITIONS OF THE PANCREAS

Surprising success has attended the efforts to control pancreatic infection, chronic as well as acute. The mortality rate in acute hemorrhagic pancreatitis has dropped from a high of around 60% to a low approximating 5% or less. One wonders whether this improvement is as real as the figures indicate. We are able to diagnose the mild, self-limited, acute infections today, and these favorable cases are correctly added to the total list for consideration of life and death, whereas 25 years ago these mild cases could not have been added to the list. In other words, the mortality rate of 25 years ago was based upon observation of the severest cases only, which would materially boost the rate. The ubiquitous use of the blood amylase, diastase, or lipase studies in suspected cases permits accurate diagnosis of mild infections. These pancreatic enzymes are elevated in most cases at some time during the early hours and days of the disease. Other conditions may cause elevation of some of these enzymes and must be known and properly appraised clinically. Diagnosis is not so difficult if there are appropriate awareness and thoughtful study of the history and results of physical examination and rather simple laboratory tests.

The first step in the mortality improvement was taken when it was realized that laparotomy was accomplishing very little in the treatment of the disease but, contrariwise, offered an unfortunate opportunity for the surgeon to manipulate inflamed abdominal organs in a serious infection, which resulted in enhancement of the fatal capacity of the disease. This was not a new concept in a

way, because competent surgeons were well aware of the danger of manipulation, particularly rough manipulation, in the presence of infection.

Today, acute pancreatitis is a surgical condition only because of its similarity to other emergency surgical conditions (notably perforated ulcer, acute intestinal obstruction, acute cholecystitis, mesenteric thrombosis, and peritonitis due to perforation of an acute appendix or diverticulum), its association with gallstones, and the cases which progress to the dreaded chronic pancreatitis. Therefore, the treatment is medical, if the diagnosis can be made, and simply consists of (1) gastric drainage, which rests the gastrointestinal tract, reduces the amount of pancreatic secretion, and lessens distention by aiding in the management of paralytic ileus; (2) antibiotic therapy, usually with a combination of two or three agents rather than with a single one; (3) antishock measures if shock is present or impending, including use of blood, blood substitutes, pressor drugs, etc.; (4) methantheline (Banthine) administration, if oral intake can be tolerated or a good parenteral preparation is available; this drug also reduces the amount of enzyme secretion, and (5) administration of calcium intravenously, as 10 cc. of 10% solution of calcium gluconate or lactate; this is a must in the severe cases because the saponifying process of the fat necrosis mechanism utilizes calcium which the body loses. Tetany may occur, even though the blood calcium determinations are at a satisfactory measured level, because of fixation to fatty acids. Intravenous calcium is harmless and can be administered at least twice daily in the severe cases without danger. We use it in the severe cases without waiting for the result of the blood calcium determination. In fact, the laboratory test is not so important, but the administration of the harmless calcium is important. These measures are continued and reduced gradually very much as in treatment of infection elsewhere in the gastrointestinal tract.

Something must be stated about what to do in case laparotomy is inadvertently performed in acute pancreatitis. As the diagnosis cannot be accurately established in all cases, some laparotomies will be performed. When the abdomen is opened, the surgeon's conduct may greatly alter the course of subsequent events. Rust-colored fluid, fat necroses, peripancreatic edema, and pancreatic enlargement and induration are quickly and easily recognized in the average patient; hence, there is no reason for prolonged, vigorous, or rough manipulation. The motto should be, "Do as little as possible and get out quickly." If gallstones and common-duct stones are present, it is usually feasible to perform cholecystotomy, cholecystectomy, or common-duct stone removal and drainage, depending upon the severity of the disease and the ease with which these procedures can be carried out. It is a matter of surgical judgment to decide which of these biliary operations can be performed with safety. This cannot be settled by a paper, speech, or panel discussion. But it is imperative to stress that it is better to have a living patient with a cholecystostomy tube and a remaining gall bladder than to have a dead patient because the surgeon wished to fulfill his desire to exploit his act to the fullest extent. All observations appear to indicate that, in this acute disease and in this one attack, cholecystostomy will be just as satisfactory as the more vigorous procedures, except perhaps when common-duct stones, particularly impacted common-duct stones, are encountered. The operation should be of short duration if biliary surgery is performed. The important matter remains: Immediately start the medical regimen outlined above.

*Chronic Pancreatitis.* — One cannot consider the management of chronic pancreatitis without bearing in mind the intimate relationship between the disease in the biliary tract and the infection in the pancreas. The incidence, association, and etiological factors are well documented in the literature. The successful control of this chronic infection largely has been had because something has been

done to alter the possibility of the commonly talked about bile-regurgitation phenomenon, i. e., the entrance of bile into the pancreatic duct, in which, if it is under pressure, infection is thought to arise. A few doubt the reality of this theory, and a newer flight from the bile-regurgitation idea claims that pancreatic-duct obstruction induces the infection. I have observed bile in the left subhepatic space in a patient undergoing acute exacerbation of a chronic pancreatitis and correctly predicted that the drainage of this bile in the upper left quadrant would be followed by pancreatic fistula and cessation of the acute attack. Colp and Doubilet [1] have observed the presence of the bile in the pancreatic duct. Retrograde visualization of this paitent's pancreatic duct showed no evidence of obstruction. It is now a fact that many cases of chronic pancreatitis do not demonstrate pancreatic-duct obstruction. Also, one wonders why the lesion which uniformly causes pancreatic-duct obstruction (carcinoma of head or body) does not cause symptomatic pancreatitis, if the pancreatic-duct-obstruction theory is correct.

I have observed two cases of aneurysm of the superior pancreaticoduodenal artery which by extrinsic pressure caused pancreatic-duct obstruction with distal stone formation and fibrosis of the distal pancreatic tissue; yet the patients demonstrated no sign of pain. The symptoms were entirely those due to bleeding from the aneurysms.

Other discarded etiologies include alcoholism, vagal stimulation, sympathetic nerve imbalance, dietary indiscretions, and psychosomatic ills. In spite of bombardment by the antagonists, bile regurgitation under pressure is still the etiology with the most promise and will apply to the majority of the cases, although as time goes on several different etiological factors may be found to exist.

Much has been learned and much has been gained, but knowledge and therapy could not be stated to be at a point of perfection. Nevertheless, our present therapeutic efforts do not deserve the spirit of futility expressed by some. We must realize that chronic pancreatitis is a serious debilitating disease, leading to drug addiction, weight loss, invalidism, social ostracism, and, when the pancreas finally becomes "burned out," to cachexia, anemia, avitaminosis, and death. Suicide has been commoner than in some other chronic diseases. Any surgical effort, regardless of its magnitude, is in order if it can offer anything logical in the control of this infection.

The therapy then will naturally flow in the channel dictated by the surgeon's etiological concept and his willingness to utilize surgery of major proportions. The next steps in the management of this perplexing problem concern the diagnosis, the selection of cases for surgical treatment, and the choice of operation.

Accurate diagnosis is often difficult, not so much because there is or is not a pathologic change in the pancreatic tissue as because of the question whether the infection in the pancreas is really causing the patient's symptoms, or whether the symptoms are the result of tremendous overlay from a more or less quiescent chronic infection in the pancreas of an unstable person. For many years pancreatic infection has been used by the profession to "name" an organic disease for actually unexplained upper abdominal distress, because physicians knew that there was no way to prove or disprove the diagnosis so conveniently but incorrectly designated. The satisfaction of the patient was of course short-lived.

Today, the diagnosis can be better established, although a long period of time and many clinical and laboratory examinations may be necessary to verify it. Also today, something very good can be done in 60% to 80% of the cases, and that is important and triumphant, if not perfect. Philosophically, let us remember that surgical treatment of peptic ulcer is not attended with 100% perfect results; yet the 92% satisfactory results in patients who were properly selected and surgically treated when all other measures have failed is indeed a worthy achievement. Medical therapy will not control chronic pancreatitis.

One must know and realize that calcified areas in the pancreas and stone formation in the ducts may be present without any symptoms of pancreatitis. These calcified areas and stones may result from chronic low-grade pancreatic infection which has not and will not always go on to the more serious subsequent events. Common sense directs that therapy should be withheld in these cases until the observers are convinced that the pain, postprandial distress, weight loss, fever, distention, constipation, and invalidism are really due to the infection in the pancreas. In other words, the microscopic pathologic picture does not always parallel the symptomatology. Stone removal per se has offered slight immediate improvement, but no permanent control of the pancreatitis. The stones in the aneurysm cases caused no symptoms; these patients would not have known of the stones or pancreatic fibrosis if they had not experienced hemorrhage from the aneurysm.

Therefore, the selection of the case for operation is probably one of the most important factors if success is to be obtained surgically in the treatment of this disease.

The therapy must vary as the symptoms and pathology change in the pancreas. The following scheme may prove helpful:

1. When gallstones are present with or without common-duct stones and there is induration in the head of the pancreas, perform a cholecystectomy, explore the common duct, and remove the stones, if present. Then the sphincter of Oddi should be dilated and a T-tube inserted which does not enter the duodenum and is permitted to remain in place for a period of at least three months, if symptoms or findings of pancreatitis accompany the gallstone symptoms. This minimal induration in the head has been studied on biopsy occasionally, and round-cell infiltration and fibrosis have been noted. Technically, the condition is a localized pancreatitis. But proper biliary surgery will satisfactorily control the majority of these cases. It is in this group that the advocates of sphincterotomy have reported excellent results. However, other surgeons have noted perhaps these same good results with gallbladder surgery only and therein exists the difficulty of properly estimating the real worth of the sphincterotomy operation.

2. When biliary tract surgery has been properly performed previously or x-ray examination has accurately shown no evidence of stones or disease in the biliary tract and the attacks of pancreatitis recur frequently enough to convince the professional observers that these attacks will continue, the most popular operation is the transduodenal cutting of the muscular fiber of the sphincter of Oddi, the operation commonly referred to as sphincterotomy.[2] The purpose of the operation is to eliminate any chance of obstruction at the ampulla of Vater, which can induce bile regurgitation when there is or is not a common channel. This operation is believed to be successful in about 60% of the cases, although for reasons expressed above this figure is by no means an accurate one. The number of successes may be much less. The second commonest operation is some form of biliary intestinal anastomosis, the purpose of which is to shunt the bile away from the ampulla in such a fashion that it cannot enter the pancreatic duct. We have used choledochojejunostomy en Roux-Y * in 14 cases since 1948 without a death, with successful control of the disease in 13 and great benefit to the 14th, who has been classified as a failure. Poth and Wolma,[7] Barclay,[8] Allbritten,[9] Faxon,† Trimble,‡ and others have achieved successes by this operation. Rousselot and coworkers [10] failed to achieve successful results in five pilot cases, although their selection of cases for the operation does not meet the requirements for selection as we have employed them. But it would be safe to say that 80% or more of the cases treated by choledochojejunostomy have been controlled and a higher percentage will certainly benefit, if careful selection of the cases is obeyed.

* References 3-6.

† Faxon, H. H.: Personal communication to the author.

‡ Trimble, I. R.: Personal communication to the author.

Some may object to the tedious procrastination we display in selecting the cases properly, but the same caution must be observed in peptic ulcer surgery—if the patients who receive surgery for peptic ulcer are not carefully and studiously selected, inferior results are obtained.

A few employ transplantation of the pancreatic duct and thereby avoid bile regurgitation.[11] No reports of the actual control rate are available, but the procedure should invoke control. It is believed by many that the operation is attended with great danger because of the chance of pancreatic necrosis or fistula and is no less extensive than the biliary enteroanastomosis. The advocates claim they are also obviating any obstruction to the pancreatic duct. Actually, the virtue of the operation, like that of sphincterotomy, may be the prevention of bile regurgitation in the pancreatic duct.

Longmire,§ originally, and DuVal,[12] later, employed caudal pancreaticojejunostomy en Roux-Y. DuVal[12] particularly uses this procedure only when he proves the presence of pancreatic-duct obstruction. He reports two successful cases with short follow-up periods. Longmire's case, rare in some other respects, failed to receive benefit. It must be emphasized that caudal pancreaticojejunostomy not only relieves pancreatic-duct obstruction but provides a passage for the regurgitated bile to go through the pancreatic-duct system so that it cannot accumulate in the duct under pressure, which is believed to be necessary to invoke the infection. This operation is fairly simple and does not require the undesirable transection of the common duct. It is to be hoped, therefore, that pancreaticojejunostomy will prove to be the safest and most successful procedure.

The permanent patency of the anastomosis is difficult to achieve in animals, which may not be the case in dealing with the larger pancreatic duct of human beings.[13]

Sympathectomy, vagotomy, and gastric resection have permitted slight, temporary benefit. These procedures rest the infected pancreas and diminish the output of pancreatic enzymes but have not offered control of the disease and have accordingly been abandoned by most of the early advocates.

*Pseudocysts.*—These may appear from trauma as well as infection. Excision is the treatment of choice if possible, i. e., when the cysts are not too large, with location in the body and tail. If excision is impossible, cystoenterostomy en Roux-Y or cystogastrostomy, depending upon the location of the cyst, is the accepted procedure. Marsupialization should be used only when the other two methods are not applicable.

*Burned-Out Pancreas.*—This is the end-stage of pancreatitis and is characterized by pain (less severe than earlier in the course of the infection), cachexia, loss of appetite, mild diabetes, and steatorrhea. Therapy ordinarily should be employed before the burned-out state is reached. There is no good surgical therapy for the seriously burned-out pancreas. Some favor partial pancreatectomy and a few total pancreatectomy.

Sphincterotomy has not been beneficial. Choledochojejunostomy cannot be safely used because the loss of neutral pancreatic juice as well as bile would very likely induce serious duodenal ulcer and would probably not favorably alter the course of the disease. Sympathectomy will alleviate some of the pain but does not add to control of the disease. We believe that a patient with a burned-out pancreas has already undergone physiological pancreatectomy but not quite a total one, and his undesirable state may be preferable to that of the totally pancreatectomized patient.

### NEOPLASMS OF THE PANCREAS

Neoplasms of the pancreas are easier to record because what can or cannot be done and the results obtained may be more accurately measured than is the case with the procedures employed in the treatment of chronic pancreatitis.

§ Longmire, W. P., in discussion on Sensenig and Bowers.[13]

*Benign Tumors.*—Islet-cell adenoma leads to lowered blood sugar levels and all the dangers which attend hyperinsulinism. The diagnosis requires awareness and a period of time in which to juggle the sugar intake against the response, along with blood sugar and dextrose-tolerance tests. Surgical removal of the tumor is attended with perfect recovery, except in the few instances when multiple tumors are encountered. When the tumor cannot be located at the operation, resection of the distal three-quarters of the pancreas should be performed. This rule is based on the fact that the majority of these adenomas are localized in the distal three-quarters of the pancreas. If subsequent operation is necessary and the tumor after careful and skillful search cannot be obtained, total pancreatectomy may be necessary.

*Cystadenoma.*—Cystadenoma is a true cystic tumor which differs from other cysts by the presence of papillomatous-looking projections from the cyst wall and its inclination to eventual malignancy.[14] Obviously, early complete removal is the only correct procedure. When one is treating a cystic lesion of the pancreas with one of the noneradicating operations, he must be certain that the cyst is not a cystadenoma.

*Malignant Tumors.*—Carcinoma in the head of the pancreas is the commonest malignant tumor of the pancreas and is followed in frequency by carcinomas located in the body and then in the tail of the organ. Successful operations can rarely be employed for those in the body and tail because symptoms appear usually when the tumor has hopelessly invaded the surrounding tissues and are indefinite even when they manifest themselves. Yet radical resection of these parts of the pancreas can be more simply and safely performed. There is urgent need for a method of detection of tumors in these localities.

Carcinoma in the head of the pancreas, because of its origin near the ampulla of Vater, initiates jaundice early in its course, which heralds its presence. It causes obstructive jaundice, with slight pain, no evidences of infection, and an enlarged gall bladder. The jaundice progresses and deepens. The diagnosis is usually not difficult but actually depends upon the findings at operation, as causes of obstructive jaundice cannot always be established by present clinical and laboratory studies. The decision to operate is easy because it is based on establishing the obstructive nature of the jaundice, which will not favorably and permanently respond to medical therapy. Differentiation at operation is difficult and cannot be perfectly accomplished, but the degree of accuracy is high in the experienced surgeon's hands. There are two types of operation: the radical pancreaticoduodenectomy (the so-called Whipple procedure) [15] and the palliative procedures, cholecystoenterostomy or gastrostomy, with variations of the shunt according to choice of different operators. At best, the operable case (in which the growth is freely movable, small, noninvasive, and nonmetastatic) should receive the radical procedure. In good hands it is attended with no higher mortality rates than the palliative procedures are. Not many five-year cures are reported after pancreaticoduodenectomy, but a longer life is certainly achieved. The average survival following palliative operations is in the neighborhood of 5 months, whereas the average survival after the radical procedure is 15 months.[16] Moreover, the patients are, so far as food intake is concerned, more normal. More important, however, would be the disastrous denial of a chance for real cure in carcinoma of the ampulla of Vater if radical pancreatectomy is never performed. It is often impossible to distinguish the ampullary carcinomas from some of the carcinomas in the head. There are many reported five-year cures in cases of ampullary tumors following the use of pancreaticoduodenectomy. Of the small number of patients with ampullary tumors seen at Kennedy Hospital, one is living without demonstrable disease at seven and a half years and another at four and a half years after operation.

One can state that the radical operation should be employed in good hands when the institution is properly equipped with labora-

tory, anesthesiological, roentgenological, and nursing facilities for management of major surgery of this type. The mortality rate in properly selected cases should not exceed 15%.

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# Evaluation of Cholecystostomy

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Cholecystostomy is a valuable surgical procedure employed by every surgeon. However, the indications for its use and the postoperative management of these patients are not generally agreed upon.* This disagreement may be concisely stated as follows:

(*a*) "All cholecystostomies, where possible, should be followed by a cholecystectomy."

(*b*) "Seventy per cent of cholecystostomy patients will remain symptom-free and require no further surgery."

These two points of view are obviously markedly divergent. The study here presented was prompted by two considerations: (1) an unusual opportunity to study a large series of cholecystostomies, and (2) a desire to evaluate this disagreement with the hope that some contribution to the problem will be made.

## BRIEF HISTORICAL REVIEW

Apparently the first cholecystostomy was performed by Jean-Louis Petit in 1743. He carefully planned the operation, removed stones from the gall bladder, and drained it, but the patient died. Bobbs, in 1867, an American, did the first cholecystostomy in one stage. However, the first really successful cholecystostomy planned and performed as such was probably done by Kocher in 1878. The operation was then repeated by many others, at an early stage predominantly by Lawson, Tait, Keen, and others. Many contributions to the early surgery of the gall bladder were made by Kehr, Moynihan, the Mayo brothers, Deaver, and others.

Prior to 1915, cholecystostomy was a commonly performed operation for both acute and chronic gall-bladder disease, probably far more frequently done than cholecystectomy. As surgical techniques improved, however, cholecystectomy became the operation of choice for chronically diseased gall bladders. Cholecystostomy was reserved for the treatment of cholecystitis.

Between the years 1916 and 1932, Heuer † advocated cholecystectomy for acute cholecystitis and suggested that cholecystostomy be reserved only for special circumstances. His work was based on an observation in 1911 that a patient in whom a cholecystectomy was performed for acute cholecystitis by Halsted had a remarkably smooth convalescence. He continued his work in Cincinnati in 1922 with essentially the same results. In 1932 at the New York Hospital, cholecystectomy for acute cholecystitis was established as policy. The results were gratifying. The mortality rate for acute cholecystitis fell to 2.46%, the lowest ever reported. In 1934, Heuer reviewed 1066 cases taken from the literature on acute cholecystitis and found the average mortality rate to be 8%, varying from a low of 4.7% to a high of 22.5%. Moreover, in 502 cases of gangrene with perforation the mortality rate was 46%, varying from a low of 15% to a high of 65%. His studies of the causes of death in acute

Received for publication Feb. 18, 1955.

Read before the New England Surgical Society meeting, Bretton Woods, N. H., Oct. 8, 1954.

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This study was made possible by funds provided by the Women's Auxiliary of the New Britain General Hospital.

* References 1, 2, and 3.

† References 4 to 7.

cholecystitis led him to estimate that early operation would eliminate 10% of the deaths from this disease and to advocate cholecystectomy as the operation of choice where feasible. Glenn,‡ in 1946, reviewed 527 cases of acute cholecystitis operated upon at the New York Hospital, of which 460 were cholecystectomies and 67 were cholecystostomies. The mortality rate over age 50, regardless of operation, was 5.4%, below 50, 1.13%. On comparison of these figures with the over-all review of the literature, it is clearly shown that early operation is the procedure of choice in acute cholecystitis and that cholecystectomy is the preferred operation.

This work is briefly reviewed because it had a wide influence and is largely responsible for the present-day swing away from cholecystostomy in acute cholecystitis and the subsequent decrease in the number of cholecystostomies performed generally.

Other arguments in favor of cholecystectomy include that of Deaver, who, as early as 1924, stated that cholecystectomy prevents secondary disease and further difficulties found to be common after cholecystostomy. In 1926, Cave [11] stated that 50% of cholecystostomies came to secondary operation, but he still preferred cholecystostomy in acute cholecystitis. Spurling and Whitaker,[12] in 1927, studied 12 patients who had had cholecystostomy performed. None had a normal gall bladder postoperatively, and four had recurrent calculi. These authors concluded that "drainage of a diseased gall bladder with the expectation that it will regain normal function is a futile procedure and one that endangers the future health of the patient." In 1928, Moore substantiated these findings in experiments on dogs. After cholecystostomy, even in the absence of disease, normal function was not regained. By 1940 it would seem that most surgeons definitely accepted Heuer's doctrine that (1) early operation in acute cholecystitis was indicated; (2) cholecystectomy where feasible was preferable to cholecystostomy; (3) when cholecystostomy was done, it should be followed in most cases by an elective cholecystectomy. Zollinger and Cutler,§ in 1943, gave the indications for cholecystostomy as follows: (1) elderly patients or those accounted poor surgical risks who present a well-defined mass; (2) seriously ill patients for whom minimal surgery is desirable; (3) presence of an abscess about the gall bladder; (4) technical difficulties preventing cholecystectomy. They advised cholecystectomy within three weeks after cholecystostomy except in the poor-risk or elderly patient.

A study of this subject by Foss [13] in 1942 presented some interesting findings. He reviewed 149 cases, of which 100 (66.6%) had cholecystostomies and 28 (18.6%) had cholecystectomies (the others had multiple operations). In those with cholecystostomy, he found the average period of relief to be 3.7 years. The average interval between the first and the second operation was 8.1 years. Of the cholecystostomy cases, 58% had stones at the second operation. He found that 20% of patients had no relief and 80% some relief. The average duration of symptoms of the first operation in the former group was 4.5 years, in the latter 3 years. The time elapsed between the first and the second operation in the former was 4 years 5 months, and in the latter 7 years 7 months. Fifteen of the 100 patients developed a biliary fistula; 7 patients developed carcinoma. The mortality rate for cholecystectomies following previous cholecystostomies was 1.5%. He concluded that secondary operations became necessary chiefly because (1) a drained gall bladder had been permitted to remain; (2) the common duct was seriously traumatized at the previous operation; (3) stones in the common duct had been overlooked or re-formed after the first procedure. He stated patients may live without symptoms even in the presence of lithiasis, but even so the disease is progressive and in the course of time extends to the ducts, liver, and pancreas. His conclusion was that a gall bladder once drained is never thereafter a normal gall bladder, if from adhesions alone.

‡ References 8 to 10.

§ References 13 and 14.

Certainly from the foregoing it would appear, then, that cholecystostomy is the operation to be rarely employed. Its results are uniformly poor; symptoms almost always recur, although the interval may be several years, and the gall bladder left behind remains diseased. It is a necessary operation in circumstances mentioned above, but if performed, should be followed by an elective cholecystectomy.

However, in sharp contradiction to all of this, reports have appeared showing that most patients are free of symptoms and regain a normally functioning gall bladder after cholecystostomy.

Mock and associates [16] reported a series of 10 patients, of whom, following cholecystectomy, 6 had normal x-rays and 8 were free of symptoms for 1 to 10 years. Jenkinson and Foley [17] reported 28 cases of cholecystostomy in which 19 patients had normal x-rays postoperatively, 2 had normal function but a contour defect, and 7 had a pathological gall bladder. In these two series, then, approximately 70% of the patients gave a normal response to x-ray examination after cholecystostomy and were symptom-free. Additional studies by Mock on 48 patients showed 68% to have normal x-rays after cholecystostomy. Eliason and Stevens [18] stated that cholecystectomy should be reserved for cases in which the anatomical relationship of duct and blood supply can be clearly visualized. In their series of 135 cases of acute cholecystitis, 92 had cholecystostomy (68%) with a mortality rate of 1.5% (2 deaths). Of these, only five had elective cholecystectomy within six months without symptoms. Only 16% (15 cases) had recurrence of symptoms.

Clearly, then, there is a marked discrepancy between the two points of view. Perhaps the extreme view, that all cholecystostomies (except in the poor-risk and the elderly patient) should be followed by a cholecystectomy, is too radical. Certainly if about 70% of patients regain normally functioning gall bladders after cholecystostomy and are symptom-free, this would be so. But what of the weight of evidence showing that "a gall bladder once drained is never thereafter normal"? These arguments, too, seem well substantiated.

The following study then, as previously stated, was undertaken in the hope of helping to evaluate the two points of view. It was further stimulated by the fact that one of us was observing after but three years of practice in this community that many postcholecystostomy patients were returning, because of symptoms, to have a cholecystectomy. This led to a casual review of the hospital records and, to our surprise, it was found that in the past 20 years a large number of cholecystostomies had been performed and that more cholecystostomies had been performed for chronic cholecystitis than for acute cholecystitis. Here, then, was offered a wealth of material for study, provided an adequate follow-up could be done.

## METHOD

The years 1930 to 1945 were chosen for study for three reasons: 1. Prior to 1930 the hospital records are difficult to review. 2. Since 1945, cholecystostomy has been infrequently performed. 3. A follow-up period of at least eight years seemed desirable. All charts were reviewed with careful check of the operative note and pathology report. The patients thus selected were then contacted by telephone through two young women assigned by the record librarian. Each was told that "this is the hospital calling," so that no individual's name came to the patient's attention. The following questions were then asked and the complete answers taken down in shorthand.

1. Have you had any further gall-bladder attacks?

2. Do you have any food intolerance?

3. Do you have any gas? Belching? Flatus?

4. Are you constipated?

5. Do you have any abdominal pain? Shoulder or back pain?

6. Have you been jaundiced?

7. Has your wound healed? Any drainage from it?

8. Have you had any further surgery?

Obviously these questions were enlarged upon when necessary. The patient was considered to be symptom-free only if the answers to all the questions were negative. The only exceptions were questions 3 and 4. Here, if belching or flatus was excessive and accompanied by constipation, gall-

bladder disease was presumed. Either manifestation alone was not considered as indicating gall-bladder disease. It was a pleasant discovery to see that most patients enjoyed being interviewed and cooperated extremely well.

## RESULTS

The results of the study are presented in tabulated form with but brief comment. The series comprised a total of 221 cases—the largest yet reported.

| | | |
|---|---|---|
| Cholecystostomy (total picture) | | 221 |
| Acute cholecystitis | 89 | |
| Chronic cholecystitis | 132 | |
| Postoperative deaths | 26 | |
| No follow-up | 51 | |
| Cases available for study | 144 | |

Attention is turned first to acute gall-bladder disease as follows:

| | | |
|---|---|---|
| Acute gall-bladder disease | | 89 |
| Postoperative deaths | 15 | |
| No follow-up | 18 | |
| Subsequent deaths* | 17 | |
| Available for study | 53 | |
| Postoperative results | | |
| Free of further complaint | 31 | (59.6%) |
| Continued symptoms | 17 | (32.7%) |
| Developed carcinoma of gall bladder | 4 | (7.7%) |

* 13 of which were followed and included in total 53.

Of this group three had further surgery: one cholecystectomy for recurrent disease; one cholecystectomy for biliary fistula; one a second cholecystostomy. There were no perforations of the gall bladder in the patients who had continued disease. The average period between first and second operations in this small group is 10.6 years. Among the 31 free of symptoms, 6 had perforations of the gall bladder. There were no postoperative biliary fistulas and no postoperative hernias.

The figures for cholecystostomy in chronic gall-bladder disease follow:

| | | |
|---|---|---|
| Chronic gall-bladder disease | | 132 |
| Postoperative deaths | 11 | |
| No follow-up | 29 | |
| Subsequent deaths* | 15 | |
| Available for study | 91 | |
| Postoperative results | | |
| Free of further complaint | 43 | (46.2%) |
| Continued symptoms | 49 | (52.7%) |
| Developed carcinoma of gall bladder | 1 | (1.1%) |

* 1 of which could be followed accurately and included in total.

Of this group who had continued symptoms (49 cases), 18 had further surgery. Of these, 17 had eventual cholecystectomy, though 5 of this group had a second cholecystostomy before coming to eventual cholecystectomy. One had a second cholecystostomy and no further surgery as yet, though he continues to have symptoms of gall-bladder disease.

The average period between first and second operations was 5.7 years, the shortest being 2 months and the longest 20 years. One case is interesting in that three operations were performed: the original cholecystostomy in 1916, a second cholecystostomy in 1930, after 12 years of symptoms, and a third cholecystostomy in 1934. The patient at present continues to have symptoms and has x-ray evidence of lithiasis.

| | | |
|---|---|---|
| Postoperative mortality and complications | | |
| Deaths in acute cholecystitis | 15 | (16.7%) |
| Deaths in chronic cholecystitis | 11 | (8.3%) |
| Over-all mortality rate for cholecystostomy | 26 | (11.8%) |
| Biliary fistulas in acute cholecystitis | 1 | (1.9%) |
| Biliary fistulas in chronic cholecystitis | 5 | (5.4%) |
| Total cases | 6 | (4.1%) |
| Postoperative incisional hernia in acute cholecystitis | 0 | |
| Postoperative incisional hernia in chronic cholecystitis | 3 | (3.2%) |
| Total cases | 3 | (2.1%) |
| Subsequently developed carcinoma in acute cholecystitis | 4 | (7.7%) |
| Subsequently developed carcinoma in chronic cholecystitis | 1 | (1.1%) |
| Total cases | 5 | (3.4%) |

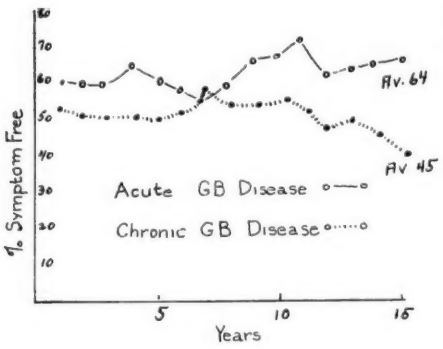
## COMMENT

The figures obtained on our study were surprising, especially since both of us have been taught that cholecystectomy, with but few exceptions, should follow every cholecystostomy. But these findings have been carefully collected and studied, and, as shown, approximately 60% of patients having a cholecystostomy for acute cholecystitis and 45% of those for chronic gall-bladder disease remain symptom-free. The 60% figure essentially substantiates the findings of Mock, Jenkinson and Foley, and Eliason and Stevens. The 45% figure for chronic disease is well below their figure of 70% but well above the view of the other extreme. Did we fail to consider some factor or factors? The only possibility seemed to be that the follow-up was not long enough. In Foss' series, the average period of relief was only 3.7 years but the interval between the first and the second operation was 8.1 years. In

our series one interval was as long as 20 years. Perhaps, then, if the figures were broken down into five three-year periods and these results studied separately, there might be a difference in recurrence of symptoms in the oldest group and the youngest. Accordingly, this was done and the findings are presented in the graph.

As can be seen from the graph, there is little change in the original finding despite differences in elapsed time. An average of 64% of the patients who had a cholecystostomy for acute gall-gladder disease remain symptom-free, and the graph approximates a straight line over the 15-year period.

In chronic gall-bladder disease the findings may be significant though the group is small.




Here the line dips gradually downward and drops off in the 12- to 15-year period, starting with a high of 55% and falling to 35%. This would seem to indicate that, as time passes, more and more patients who have had cholecystostomy for chronic cholecystitis will develop symptoms.

Our observations from this study are as follows:

1. Approximately 60% to 65% of patients having a cholecystostomy for acute gall-bladder disease remained symptom-free.

2. Of patients having cholecystostomy for chronic gall-bladder disease, 45% remained symptom-free for a while. However, as time passed, more and more of these patients showed evidence of gall-bladder disease.

3. It would seem that cholecystostomy alone is not the poor procedure in acute cholecystitis that some authors indicate.

4. It cannot be considered a good elective procedure, however, if 35% of patients develop recurrence of symptoms and gall-bladder disease necessitating further surgery.

5. Cholecystostomy is definitely a poor procedure in chronic cholecystitis.

Our conclusions from these observations are the following:

1. Cholecystectomy should always be performed where possible.

2. Cholecystectomy following cholecystostomy for acute cholecystitis would seem to be indicated only in those patients who subsequently develop gall-bladder disease. The patient may fall into the 65% group that remains free of symptoms and presumably of disease as well.

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# Paget's Disease of the Nipple

## Review of Twenty-Five Cases with Special Reference to Melanin Pigmentation of "Paget Cells"

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Although it has been pointed out that some lesions diagnosed as extramammary Paget's disease may in reality be superficial malignant melanomas,[9] it is apparently not generally appreciated that mammary Paget's disease may also simulate malignant melanoma in its histologic appearance. We were confronted with this diagnostic problem when we recently encountered Case 22 of the present series. The patient's complaint was itching of the nipple, and the nipple seemed firmer and more deeply pigmented than normal. It did not have the eczematoid appearance commonly seen in Paget's disease. The problem was subsequently resolved by the demonstration, on serial sectioning of the subareolar area, of a tiny focus of intraductal carcinoma and by the appearance within two weeks of a second, somewhat similar case. In the latter the histologic appearance of the nipple was nearly identical with that of the first case, although the nipple was crusted. This lesion was associated with frank mammary cancer. The finding of pigment in the so-called Paget cells and the necessity of carrying out serial sectioning to demonstrate the associated breast carcinoma in one case of Paget's disease of the nipple prompted us to review the experience with this disease at the Hospital of the University of Pennsylvania.

Submitted for publication May 23, 1955.

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### MATERIAL

Twenty-five cases of Paget's disease of the nipple, diagnosed in the period 1942-1954, were found in the files of the laboratory of surgical pathology. The clinical records of these cases, as well as the pathologic material, were completely restudied, special attention being given to the presence or absence of pigment in the Paget cells, the presence or absence of a palpable mammary tumor, the extent of the associated mammary carcinoma, the status of the axillary lymph nodes, and the end-results.

In those cases in which pigment granules were seen in the Paget cells, special stains were carried out for melanin (ammoniacal silver nitrate reduction), iron (Turnbull's blue reaction), lipoids (Sudan IV), and acid-fastness. In addition, mucicarmine stains were done in all the cases.

A summary of pertinent clinical and pathologic data is presented in Table 1.

### CLINICAL ASPECTS

Five patients complained initially of a mass in the breast, and a sixth complained of an ulcer in the inframammary fold. The presenting complaint of the remaining 19 referred directly to the nipple. In 6 of the 19, the nipple was described as ulcerated; in 6 others it was described as "scaly," "reddened," or showing an "eczematoid eruption." Four patients listed bloody nipple discharge as their presenting complaint. (The nipple of one of these patients appeared normal grossly, and one was abnormal only in being tender. The other two

presented characteristic crusted excoriations.) Two of the nineteen patients complained primarily of itching of the nipple. (The nipple of one showed typical excoriation. The nipple of the other was described as firm and deeply pigmented.)

The 19th patient (Case 16) is of particular interest. This patient had suffered from psoriasis for many years. Eighteen months prior to being seen in this hospital she noted the presence of a nipple eruption similar to her chronic psoriatic lesions. This was treated

TABLE 1.—*Summary of Clinical and Pathologic Data in Paget's Disease of the Nipple*

| Case No. | Age | Race | Complaint | Preoperative Duration, Mo. | Description of Nipple | Palpable Mammary Tumor | Pigment in Paget Cells | Character of Breast Tumor | Axillary Lymph Node Metastases | Follow-Up |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 67 | N | Ulceration of nipple | 12 | Ulcerated | + | + | Intraductal | 0 | Local recurrence excised 8 yr. postoperatively; living and well 12 yr. |
| 2 | 56 | W | Breast mass | "Few" | Firm | + | 0 | Infiltrating | + | Died 9 mo. |
| 3 | 45 | W | Breast mass | 17 | Normal | + | 0 | Infiltrating | + | Died 4 yr. |
| 4 | 55 | N | Nipple ulceration | 13 | Ulcerated | + | + | Infiltrating | + | Living and well 10 yr. |
| 5 | 52 | W | Breast mass | 12 | Ulcerated | + | 0 | Intraductal | 0 | Living and well 11 yr. |
| 6 | 43 | W | Itching of nipple | 12 | Excoriated and scaly | + | 0 | Infiltrating | + | Died 3 yr. |
| 7* | 50 | W | Itching and redness of nipple | 60 | ..... | + | + | Infiltrating | + | Died 2 yr. |
| 8 | 50 | W | Scaly nipple | 12 | Scaly lesion | 0 | + | Nipple biopsy only; definitive treatment elsewhere | .. | Living and well 8 yr. |
| 9 | 47 | W | Discharge from nipple | 2 | Eczematoid | 0 | 0 | Intraductal | 0 | Living and well 8 yr. |
| 10 | 53 | W | Eczematoid eruption | 3 | Ulceration of nipple | 0 | 0 | Intraductal | 0 | Living and well 5 yr. |
| 11 | 51 | W | Ulceration of nipple | 12 | Scaling lesion | 0 | 0 | Intraductal | 0 | Living and well 5 yr. |
| 12 | 68 | W | Bloody nipple discharge | 2 | Normal | + | + | Infiltrating | + | Died 16 mo. |
| 13 | 44 | W | Ulceration of nipple | 48 | Red, denuded area including whole areola | 0 | 0 | Infiltrating | + | Died 6 yr. |
| 14 | 50 | W | Milky nipple discharge | 36 | Tender | 0 | 0 | Intraductal | 0 | Living and well 4 yr. |
| 15 | 78 | W | Red, eczematous nipple | 12 | Red, scaly area including entire areola | 0 | 0 | Intraductal | Simple mastectomy | Living 4 yr., hemiplegic |
| 16 | 39 | W | Patient had psoriasis; nipple lesion thought to be the same | 18 | Crusted, erythematous | 0 | 0 | Intraductal | 0 | Living and well 2 yr. |
| 17 | 74 | W | Ulceration of nipple | 2 | Ulcerated | 0 | 0 | Intraductal | 0 | Living and well 2 yr. |
| 18 | 71 | W | Crusted nipple | "Several" | Crusted | + | 0 | Infiltrating | 0 | Living and well 2 yr. |
| 19 | 44 | W | Breast mass, nipple discharge | 120 | Excoriated | + | + | Infiltrating | 0 | Living and well 2 yr. |
| 20 | 40 | W | Ulceration of nipple | 8 | Crusted desquamated | 0 | + | Intraductal | 0 | Living and well 1 yr. |
| 21 | 69 | W | Ulcer beneath breast | 2 | Normal | + | 0 | Infiltrating | + | Living and well 6 mo. |
| 22 | 72 | W | Itching of nipple | 2 | Firm, deeply pigmented | 0 | + | Intraductal | 0 | Living and well 4 mo. |
| 23 | 47 | N | Nipple "lesion" | 1 | Tender, crusted | 0 | + | Infiltrating | 0 | Living and well 2 mo. |
| 24 | 65 | N | Pain and bloody nipple discharge | 2 | Tender, crusted | 0 | + | Intraductal | 0 | Living and well 5 mo. |
| 25 | 53 | W | Breast mass. | Found on routine exam. | Normal | + | 0 | Intraductal | 0 | Living and well 5 mo. |

* One year elapsed between nipple biopsy elsewhere and radical masteetomy performed at the Hospital of the University of Pennsylvania.

TABLE 2.—*Presenting Complaint in Paget's Disease of the Nipple*

| | |
|---|---|
| Tumor mass in breast | 5 |
| Ulcer beneath breast | 1 |
| Ulceration of nipple | 6 |
| Eczematoid eruption, etc., of nipple | 6 |
| Discharge from nipple | 4 |
| Itching of the nipple | 2 |
| Psoriasis | 1 |
| Total | 25 |

with various local medicaments, but when the lesion failed to resolve suspicion was finally aroused and biopsy recommended (Table 2).

Of the patients whose primary complaints were referred to the breast rather than the nipple, three had grossly normal nipples and one nipple was abnormal only in being firm. Added to the three cases in which the presenting complaint was itching of or discharge from the nipple despite a normal or near-normal clinical appearance, this makes a total of seven cases in which the nipple lacked the gross characteristics usually associated with Paget's disease. This is consistent with the observation of Dockerty and Harrington,[4] who reported seven instances of histologically typical Paget's disease in sections of grossly normal-appearing nipples. They state that the bulky underlying breast carcinomas in these cases could hardly be considered secondary to such "preclinical" nipple lesions, and they believe such cases to constitute a strong argument against considering Paget's disease as a primary skin lesion with secondary manifestations in the breast.

A total of five patients had a nipple discharge, which was bloody in four. Only one of these patients had a palpable tumor, but this patient, as well as two others, had reasonably typical-appearing gross lesions.

A tumor mass was palpable in the breast in 12 (48%) of the patients in this series. This figure is of the same order of magnitude as the 59% reported by Costello[3] and the 39% reported by Colcock and Sommers.[2] The prognostic significance of the presence or absence of a palpable mass will be discussed later.

### PATHOLOGIC FINDINGS

A distinct mammary carcinoma was found in each of the 24 cases in this series in which appropriate material was available for study. In one case it was necessary to make serial sections of the subareolar area (Fig. 1) in order to demonstrate it, and three other tumors were so small as to require examination of multiple sections from each of serial blocks for their demonstration.

Thirteen of the tumors were confined to the ducts, insofar as could be determined by our examination. Generally this involved study of more sections than the usual routine, but, except in such special cases as those referred to above, serial or step sectioning

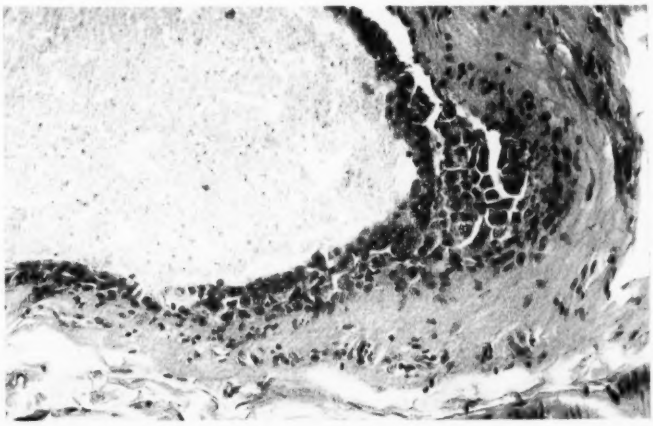

Fig. 1 (Case 22).—Focus of intraductal carcinoma found beneath areola on serial sectioning; × 195.

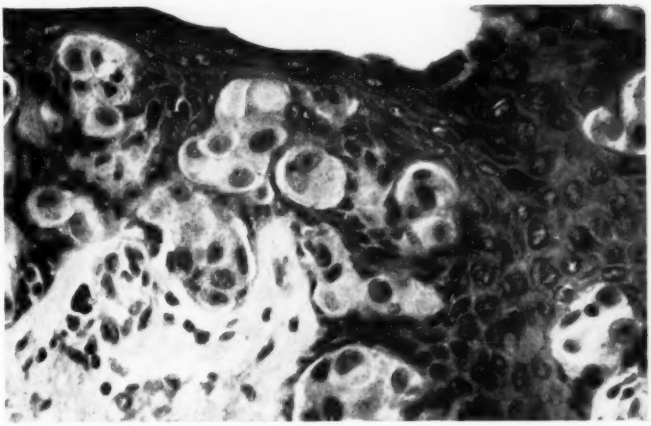

Fig. 2 (Case 22). — Granules of melanin pigment in so-called "Paget cells"; × 480.

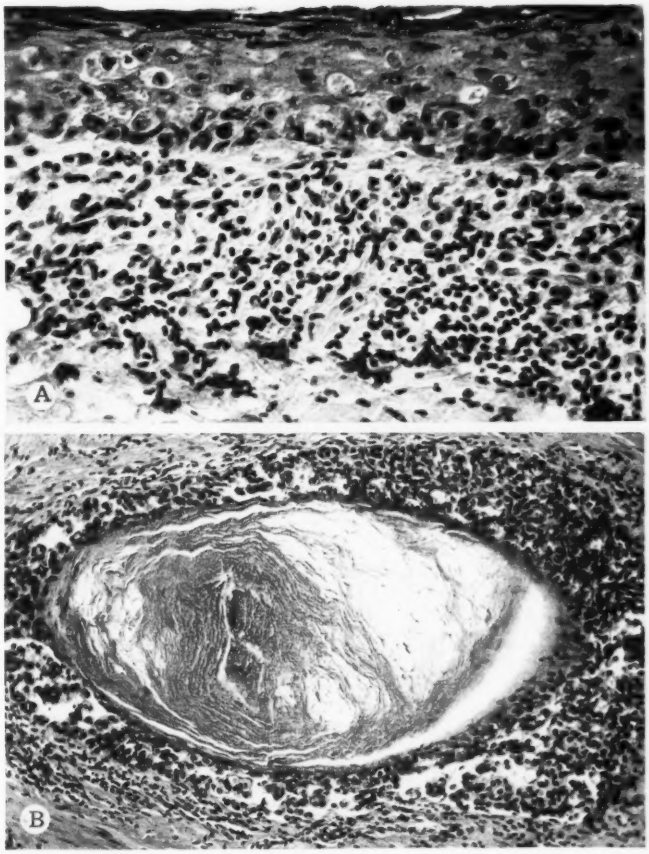


Fig. 3 (Case 23).—*A*, Paget's disease of the nipple. Pigment is present in the so-called "Paget cells" but is particularly heavy in the dermis; × 375. *B*, Associated intraductal mammary carcinoma; × 150.

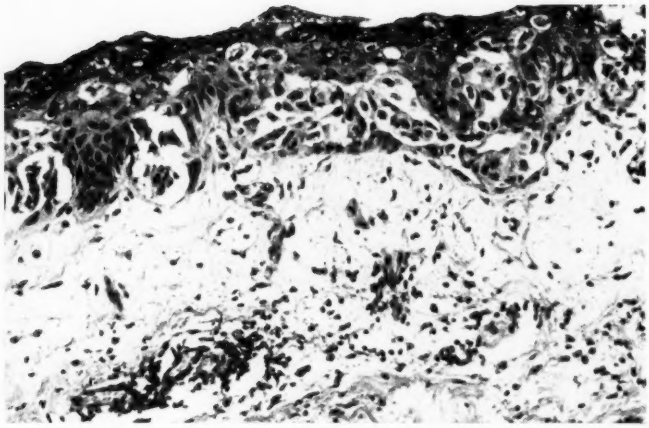

Fig. 4 (Case 7).—Biopsy of the nipple. Paget's disease simulating the histologic appearance of the junction mole or superficial melanoma; × 250.

was not carried out. It is a matter of general agreement that Paget's disease of the nipple and carcinoma of the mammary gland are regularly associated. The series of Dockerty and Pratt [5] is significant—in all their 100 cases of Paget's disease of the nipple mammary carcinoma was demonstrated.

In eight of our cases axillary lymph nodes were demonstrated to be the site of metastasis. Seven of these eight patients had clinically palpable tumors. The occurrence of axillary metastases with Paget's disease of the nipple in the absence of a mammary tumor mass is infrequently recorded, although Costello [3] noted 4 such instances in his series of 29 cases and Sarason and Prior [8] reported a similar case in a male. In our Case No. 13 axillary metastases were present without a mass being palpable in the breast. In this case the area of skin involvement was extensive, and, as in the other seven cases with metastases, stromal invasion was readily demonstrated histologically.

### PIGMENTATION

In 10 of the 25 cases comprising this series (40%), pigment granules were demonstrated within the cytoplasm of the "Paget cells." The degree of pigmentation varied widely. In some cases only an occasional cell was pigmented; in contrast were examples like Case 22 (Fig. 2), which created the original problem of differentiation from malignant melanoma, and Case 23, in which pigmentation was particularly heavy (Fig. 3). In many cases, particularly where "Paget cells" were few and the pigmentation insufficiently marked to attract attention, the resemblance to the so-called junctional features of moles and melanomas was not striking. In Case 7, however, the histologic appearance simulated almost exactly that of "junction" moles or melanomas (Fig. 4). This patient did not

Fig. 5 (Case 7).—Radical mastectomy one year after biopsy. Mammary carcinoma invading stroma and lymphatic vessels; × 150.

TABLE 3.—*Comparison of Cases of Paget's Disease of the Nipple With and Without Pigmentation of Paget Cells*

| | Cases with Pigment | Cases Without Pigment | Total |
|---|---|---|---|
| No. of cases | 10 | 15 | 25 |
| Typical nipple lesion | 8 | 10 | 18 |
| Palpable breast tumor | 4 | 8 | 12 |
| Invasive growth | 6 | 5 | 11 |
| Axillary metastases | 3 | 5 | 8 |

have a mastectomy until one year after nipple biopsy, and at that time she had an extensive carcinoma with axillary metastases (Fig. 5). The cases with pigmentation did not differ histologically from those without, except for the presence of the pigment. Likewise, there were no significant clinical differences in the two groups. Four of the 25 patients in this series were Negroes, and in all the "Paget cells" were pigmented. This group included Case 23, in which pigmentation was especially heavy. Of the 10 patients in the group with pigment, the clinical appearance of the nipple was more or less typical of Paget's disease in 8. The mammary carcinomas were growing infiltratively in six, although only four were clinically palpable. Axillary lymph-node metastases were present in three. These data are compared with corresponding figures for the cases without Paget-cell pigmentation in Table 3. In no case was pigment seen in tumor cells within the breast or in axillary lymph nodes.

The evidence at hand indicates that the pigment is melanin, similar to that of the skin. The granules consistently reduced ammoniacal silver nitrate; they did not give a reaction for iron. The pigment was not acid-fast and was not stained with Sudan IV. Unfortunately, suitable material was not available for the dopa reaction.

Dockerty and Pratt [5] demonstrated mucicarminophilic material in four cases of extramammary Paget's disease, both in the "Paget cells" in the skin and in the associated rectal or vulvar carcinoma. They considered this as further strong evidence for the metastatic nature of Paget's disease. Mucicarmine stains were done on the sections of the nipple in our cases in an attempt to repeat this for mammary Paget's disease. None of the cases was definitely positive, although one case was equivocally so.

## TREATMENT AND RESULTS

With but two exceptions the patients in this series were treated by radical mastectomy. One exception was an elderly poor-risk patient who had a simple mastectomy under local anesthesia. The other exception was treated elsewhere. In patients presenting with a nipple lesion and without a mass in the breast, the usual procedure has been to perform a biopsy and withhold definitive treatment until the diagnosis has been established on the basis of paraffin sections. In the presence of a mammary tumor mass the patient has been managed just as if no nipple lesion were present.

Colcock and Sommers [2] have reported excellent results with simple mastectomy in cases without palpable breast tumors and without demonstration of invasive growth on pathologic examination. It has seemed more prudent to us to consider radical mastectomy the treatment of choice, since there are cases, however few, in which axillary lymph-node metastases occur in the absence of a palpable breast tumor. We have also observed instances of intraductal breast cancer without Paget's disease in which axillary lymph-node metastases have been found in the absence of demonstrated stromal invasion in the breast.

Only 13 of the patients in this series were treated 5 or more years ago. Four of the five patients without palpable mammary tumors are alive and without evidence of

TABLE 4.—*Five-Year End-Results in Paget's Disease*

| | Total Cases | Cases Without Axillary Metastases | 5-Year Survivals |
|---|---|---|---|
| Without palpable mass | 5 | 4 | 4 |
| With palpable mass | 8 | 2 | 3* |
| Totals | 13 | 6 | 7 |

* One surviving patient had axillary lymph-node metastases.

disease 5 to 8 years after operation. The patient who failed to survive has already been referred to above—she had extensive skin involvement and axillary lymph-node metastases at the time of radical mastectomy. On the other hand, only three of the eight patients who presented with palpable mammary tumors have survived radical mastectomy for 5 or more years. Two of the three surviving patients did not have axillary lymph-node metastases, whereas one survivor and all those who died of the disease did. Figures for the total series, regardless of the length of follow-up, are essentially similar. Twelve of the thirteen patients without a palpable breast mass are living, whereas only 7 of the 12 patients with palpable tumors survive.

These figures emphasize what has been pointed out previously*—that the presence or absence of a palpable mammary tumor has a very important bearing on the prognosis of Paget's disease of the nipple. Those patients who have only nipple lesions on physical examination have an excellent chance of survival, whereas in the presence of a breast tumor the prognosis is that of mammary carcinoma in general.

## COMMENT

The phenomenon of Paget-cell pigmentation and the problem of differential diagnosis of Paget's disease of the nipple from malignant melanoma have received little mention in the literature. However, Allen and Spitz,[1] in their paper on malignant melanomas, state that the differentiation between the two lesions frequently must depend on the demonstration of an associated breast cancer and that lacking such a finding the differential diagnosis sometimes cannot be made with certainty. According to Masson,[6] melanin may accumulate in metastatic cancer cells within the epidermis, just as in epidermal cells.

The frequency with which pigmentation of the Paget cells was encountered in this series emphasizes the point that an especially intensive search must be made for an associated intraductal carcinoma in those cases where the diagnosis of intraepidermal malignant melanoma of the nipple is under consideration. However, it would appear that malignant melanoma, especially the superficial variety, only rarely involves the nipple. The diagram of Pack and associates showing the distribution of 1225 malignant melanomas indicates no example of such a tumor of the nipple.[7]

Although this series includes no instance in which the diagnosis of Paget's disease was made only after serial sectioning of the nipple, as was true in the cases reported by Dockerty and Harrington,[4] it does include four cases in which the nipples were grossly normal and one in which the gross nipple changes were minimal but in which the breasts contained palpable tumors. Such cases favor the belief that the nipple lesion is secondary to the breast lesion, rather than the reverse. They have no bearing on the view still maintained by some that the two lesions are coincidental only and represent quite distinct and separate neoplastic processes.

## SUMMARY

The clinical data and pathologic material of 25 cases of Paget's disease of the nipple have been reviewed. Particular attention is called to the fact that melanin pigmentation of the Paget cells is frequent (40% of this series) and may be heavy. The histologic appearance of the "junction mole," or superficial malignant melanoma, may be simulated almost perfectly.

Carcinoma of the mammary gland was demonstrated in all cases (24) in which the material was avaliable to search for it, although a very intensive search was required in four cases, involving serial sectioning of the subareolar area in one instance. In four cases the nipple appeared grossly normal, and in three the gross findings were minimal. It is considered that this experience adds further support to the interpretation of the nipple lesion as a manifestation of spread of mammary carcinoma.

Because axillary lymph-node metastases may occur in the absence of a palpable mass

* References 2 and 10.

in the breast (one case in this series), radical mastectomy is considered the treatment of choice for Paget's disease of the nipple. The end-results in this small series support the general experience that the prognosis of Paget's disease without a palpable mammary tumor is excellent, but, if a mammary tumor is present, the prognosis does not differ materially from that of mammary carcinoma in general.

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# Nonsuture Blood-Vessel Anastomosis

## An Experimental Study Using Polyethylene as the Prosthetic Material

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The techniques of vascular anastomosis and blood vessel grafting are undergoing constant revision and improvement. Developments in vascular surgery have been recently hastened by the widening scope of therapeutic applications. Almost no segment of the human aorta is immune from surgical attack at present, encompassing such various pathologic entities as coarctation, thrombosis, aneurysm, and arteriosclerosis. In many of these conditions after the diseased segment has been resected it is impossible to restore vascular continuity without the interposition of a graft. The present-day surgeon has his choice of fresh, frozen, and freeze-dried arterial homografts, or he may prefer one of the recently introduced plastic cloths for a grafting material. Grafts in the venous system are less frequently indicated than in the arterial, but they have found recent application in the performance of portacaval shunts and in treatment of the superior vena cava syndrome. Venous autografts are usually used for grafts in the venous system.

The success of any surgical procedure on the vascular system depends to some degree on the type of anastomosis performed. Since the monumental work of Carrel and Guthrie, suture anastomosis has been the principal method of restoring vascular continuity. We believed it desirable to reassess nonsuture anastomosis, because of the advantages of simplicity and speed possessed by this technique.

### HISTORICAL

In 1897 Nitze [1] presented the first work on nonsuture anastomosis, at the International Medical Congress in Moscow. He used ivory rings, through which one vascular end was drawn and turned back. The other vessel end was pulled over this prosthesis, and a ligature held the ends securely in place. In 1900 Payr * introduced a similar technique using magnesium rings, and in 1904 he proposed an improvement with a male and a female adapter, eliminating the need for ligatures. Lespinasse [4] in 1910 utilized metal flanges of magnesium in a series of nonsuture anastomoses in the carotid arteries of dogs, with excellent short-term results. In 1938 Heymans [5] reported a successful 15-year experience with brass Payr cannulae in acute experiments. All these previous procedures suffered from the disadvantage of utilizing a prosthetic material which was reactive in the tissues, thus obscuring the possible long-term adequacy of the method.

Tuffier [6] reported the use of paraffin-lined silver tubes in bridging arterial defects in battle wounds of World War II. He recognized that patency was a matter of only a few days with these tubes but believed that they would tide over a limb with a severed major vessel until adequate collateral circulation could be formed. In 1942 Blakemore † and his associates reintroduced the nonsuture method of anastomosis, using Vitallium, a

Submitted for publication June 1, 1955.

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The opinions or assertions contained herein are the private ones of the writers and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.

* References 2 and 3.

† References 7, 8, 9, and 10.

nonreactive metal, as the prosthetic material. They found that vein-lined Vitallium tubes were easy to insert in the arterial system, maintained a high incidence of patency, and were superior to suture anastomosis in the presence of infection. Human cases of arterial replacement with vein-lined Vitallium prostheses were reported, and a series of portacaval and splenorenal shunts were performed with the technique. Weiss and Lam [11] demonstrated that vein-lined tantalum tubes could be used as well as Vitallium ones, and these workers corroborated Blakemore's figure of 90% short-term patency for arterial nonsuture anastomoses. Hufnagel [12] demonstrated in 1947 that highly polished Lucite tubes would remain patent in the canine thoracic aorta, and this was the first example of long-term patency using prostheses without vein-graft linings. Donovan [13] in 1949 and Moore [14] in 1940 found that plain or siliconed polyethylene tubing became thrombosed in a high percentage of cases when inserted in dog and rabbit thoracic aortas.

The results of Blakemore's nonsuture anastomosis in the venous system were equivocal, and in 1947 Johns [15] contrasted suture and nonsuture methods of anastomosis as applied to the portal system and decided strongly in favor of the suture method. Also in 1947 Swenson and Gross [16] reported a successful series of venous anastomoses using absorbable fibrin tubes, which would allow for growth of the vein after absorption of the prosthesis. There has been no further report of this very promising work. Daniel [17] in 1952 demonstrated that Lucite tubes thrombose when inserted in the canine portal and superior mesenteric veins.

## MATERIAL AND METHODS

The present study evaluates vein-lined polyethylene tubing used in nonsuture anastomoses. The first group of experiments tested autologous vein-lined polyethylene tubing used to bridge defects in the aorta and the carotid and femoral arteries of the dog. The second set of experiments was devised to determine the most effective method of using polyethylene tubing as a venous prosthesis. In the third set the fate of venous homografts as lining materials for the polyethylene tubing was ascertained. A principal result of the investigation was the contrast between solid and perforated vein-lined polyethylene tubes.

Healthy adult mongrel dogs ranging in weight from 15 to 25 kg. were used for all experiments, and intravenous pentobarbital (Nembutal), 30 mg/kg., was used for anesthesia. All operations were performed under sterile conditions, and antibiotics were not given. Polyethylene tubing of various diameters was cut to the desired lengths and the ends slightly flared with heat to form ridges which would hold ties (Fig. 1).

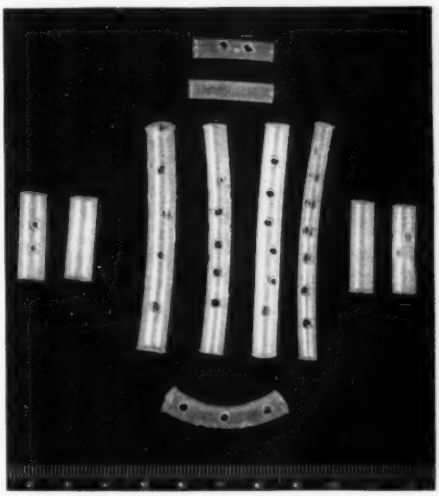


Fig. 1.—Various sizes and lengths of polyethylene tubing. Note presence of perforations in some tubes and flared ends in all tubes.

In the artery experiments, perforated polyethylene tubing 3 cm. long was used, and venous autografts were taken from the right external jugular vein. The vein segment was placed inside a polyethylene tube of appropriate diameter and the ends of the graft cuffed back over the tube. The graft was then secured tightly to the tube by a single 000 nonabsorbable surgical (silk) suture tie at each end. A 2-cm. segment of the aorta, or of the carotid or femoral arteries, was resected and the severed arterial ends triangulated with hemostats. The dog's external jugular vein has one to three valves, and particular attention must be given to the direction of flow in the grafted venous segment. The prosthesis was then inserted into the open artery ends

Fig. 2.—A 2-cm. solid vein-lined polyethylene tube in situ in external jugular vein of dog.

and tied in place with 00 nonabsorbable surgical (silk) ligatures, one at each end.

There were six parts to the experiments evaluating venous nonsuture anastomosis. In Part I a series of dogs had polyethylene tubes 2 cm. long treated with a silicone compound (GE Dri Film—SC-87) inserted into their external jugular veins. These polyethylene tubes without vein linings served as controls for the other five groups of animals and were simply tied in the recipient vein after resection of 1 cm. of its length. Parts II and III consisted of a contrast between solid and perforated vein-lined polyethylene tubes, 2 cm. long, tied in place in the external jugular vein (Fig. 2). The grafts were prepared as in the artery experiments, and, after resection of 1.5-cm. segments of the left external jugular vein, the prostheses were tied in place with single 00 nonabsorbable surgical (silk) sutures at each end. The only difference between Parts II and III was the presence of six perforations, each 2 mm. in diameter, in the polyethylene tubing used in the Part III experiments. In Part IV, two 0.5-cm. polyethylene tubes were used with 2 cm. vein grafts taken from the right external jugular vein and were inserted into the left external jugular vein. This left unsupported vein graft in the central portion of the prosthesis. Part V evaluated vein-lined 6-cm. polyethylene tubes having 18 perforations, each 2 mm. in diameter, which were tied in the left external jugular vein. A series of venous nonsuture anastomoses in the portal system were performed in Part VI. In some instances straight prostheses 2 cm. in length, lined with external-jugular-vein grafts, were tied in the portal vein. Nonsuture anastomoses were also made between the left renal and superior mesenteric vein with 3-cm. curved polyethylene tubing lined with external jugular vein grafts.

The superior vena cava was chosen for the site of prosthetic insertion for the third set of experiments, and fresh homografts were taken under sterile conditions from the superior vena cava in three instances and the external jugular vein in two. When the recipient was much smaller than the donor, a graft from the external jugular vein of the donor was used, since it was approximately the same diameter as the superior vena cava of the recipient. As in previous experiments, the graft was cuffed back and tied in place with 000 nonabsorbable surgical (silk) sutures and the prosthesis tied in the superior vena cava with 00 nonabsorbable surgical (silk) suture ties. Prostheses were 3 cm. long, and a 2-cm. segment of superior vena cava was resected. In an average (15 to 20 kg.) dog this leaves enough superior vena cava above and below to tie the prosthesis in place. In an occasional animal it was necessary to dissect the intrapericardiac superior vena cava in order adequately to insert the prosthesis.

With careful attention to proper size relationship of prosthesis to recipient vessel, insertion time for the various prostheses was very short. In readily accessible vessels, such as the external jugular vein and femoral and carotid arteries, total elapsed time from initial incision of vessel to completion of prosthetic insertion and return of blood flow through the vessel was seldom more than five minutes and often only two or three minutes. In deeper areas, such as the portal and superior vena cava veins, insertion time varied from 5 to 15 minutes.

Serial venograms were performed with 35% iodopyracet (Diodrast) in representative experiments of both Group 2 and Group 3. Vascularization of the vein-graft lining of the perforated polyethylene tubes in the external jugular vein was visualized

TABLE 1.—*Arterial Prostheses*

| Site | Tube Size * | Interval Between Insertion and Death | Result |
|---|---|---|---|
| 1. Carotid | 330 | 17 days | Excellent |
| 2. Carotid | 330 | 18 days | Thrombosis |
| 3. Femoral | 350 | 15 days | Excellent |
| 4. Femoral | 360 | 15 days | Excellent |
| 5. Aorta | 380 | 8 days | Excellent |
| 6. Aorta | 380 | 51 days | Excellent |
| 7. Aorta | 380 | 54 days | Good, slight narrowing |

* Tube sizes (inside by outside diameters)—330 (2.92 × 3.73 mm.), 350 (3.17 × 3.99 mm.), 360 (3.76 × 4.82 mm.), 380 (3.99 × 5.95 mm.), 390 (5.28 × 6.86 mm.), 410 (6.35 × 7.35 mm.), 430 (8.13 × 9.25 mm.).

by clamping the vein above and below the prosthesis in situ and injecting 10 cc. of 1:8 India ink into the common carotid artery on the side of the prosthesis. The appearance of the intima of the grafts in perforated prostheses was contrasted with that of grafts in solid prostheses with similar injections. Microscopic sections were made of tissue which had grown through the perforations of the perforated prostheses. Before necropsy of dogs containing prostheses, flow through the prosthetic area was determined by cutting the vessel below and observing flow directly.

## RESULTS

*Group 1.*—Arterial System: Venous autograft lining for prostheses. Table 1 gives results with perforated polyethylene vein-lined tubes in the arterial system. All cases classified as excellent had little or no lumen narrowing, good flow through the prosthetic area, and no thrombi on the vein-graft lining of the prosthesis. In the one carotid artery prosthesis with thrombosis, the operative protocol mentioned a twist in the vein graft at the time of insertion. Attention to detail eliminated further difficulty in arterial nonsuture anastomoses. In all experiments excellent intima to intima healing was observed between the vein graft of the prosthesis and the arterial wall. In spite of the use of only one tie at each end of the prosthesis to hold it in the vessel, there was no instance of hemorrhage or blowout.

*Group 2.*—Venous System: Venous autograft lining for prostheses. Table 2 summarizes findings in Parts I to IV of the experiments with venous-autograft-lined polyethylene tubes. A good result was taken to mean good flow through the prosthetic area, little or no fibrosis or constriction of the vein graft, and no thrombi. Any appreciable decrease of flow, fibrosis, or thrombus formation led to a classification of poor. Average follow-up for each group of experiments in Table 2 is 20 days. Size 380 and 390 polyethylene tubes were used for all experiments in these groups (footnote, Table 1). Table 3 shows the sharp contrast between the perforated and solid polyethylene vein-lined prostheses. The marked superiority of perforated over solid vein-lined polyethylene prostheses is clearly shown by these results. The intima of the vein graft was almost invariably well healed with the intima of the recipient vessel in the case of the perforated prostheses.

In a series of 16 experiments with 6-cm. perforated polyethylene vein-lined tubes (Part V), 50% good results were obtained with an average follow-up period of 18 days. These 6-cm. tubes were placed in the left external jugular vein. Thirteen prostheses placed in the portal system comprised Part VI. Of nine superior mesenteric-renal vein nonsuture anastomoses, eight were followed for an average time of 20 days. One dog died on the first postoperative day with a hemorrhagic pneumonia but patent prosthesis.

TABLE 2.—*Results: Two-Centimeter Polyethylene Tubes*

| | Good | Poor |
|---|---|---|
| Solid vein-lined (17 dogs) | 18% | 82% |
| Perforated vein-lined (23 dogs) | 78% | 22% |
| Two-tube vein-lined (13 dogs) | 77% | 23% |
| Siliconed (10 dogs) | .... | 100% |

TABLE 3.—*Comparison: Two-Centimeter Solid and Two-Centimeter Perforated Vein-Lined Polyethylene Tubes*

| | Solid | | Perforated | |
|---|---|---|---|---|
| Follow-Up, Weeks | Good | Poor | Good | Poor |
| 1 to 2 | 2 | 3 | 5 | 2 |
| 2 to 4 | 1 | 11 | 12 | 4 |

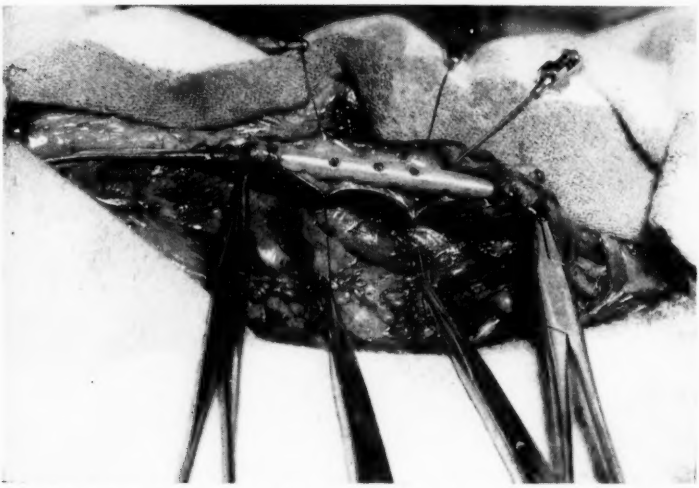


Fig. 3.—Dissection of a 6-cm. vein-lined perforated polyethylene tube in external jugular vein of dog for 35 days. Note tissue passing through perforations.

Fibrosis and thickening of the vein grafts was much more marked in the portal than in the external jugular system of veins. Perforated 2- and 6-cm. vein-lined prostheses in the external jugular vein were followed for as long as two months, with noticeable fibrosis in only a small percentage. However, more than 50% of the prostheses in the portal circuit which remained patent demonstrated considerable narrowing of the vein graft in the polyethylene tubing.

Dissection of the perforated tubes in situ before necropsy revealed highly vascular bits of tissue bridging from the peritubal areas through the perforations to the vein-graft lining of the prosthesis (Fig. 3). Photomicrographs of the tissue which grew through the perforations revealed numerous

Fig. 4.—Venogram of a 6-cm. perforated polyethylene vein-lined tube in situ for 32 days. Note absence of collateral venous circulation. Channel through prosthesis is of same diameter as immediately postoperatively.

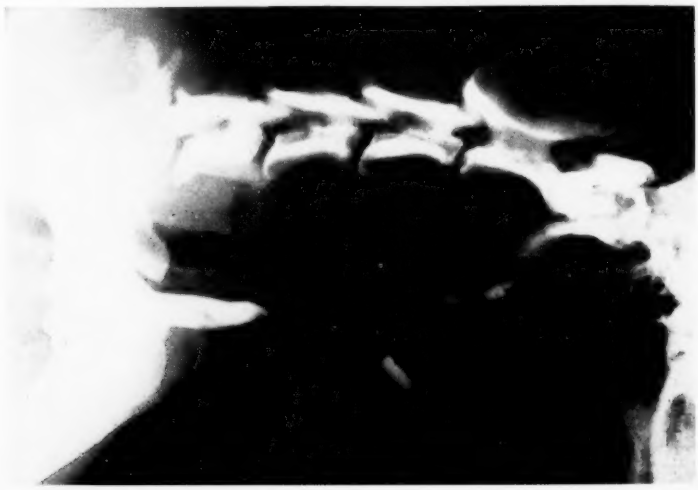
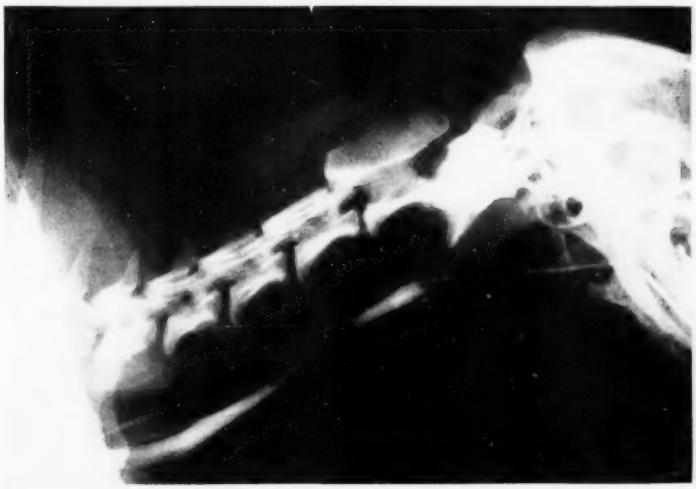


Fig. 5.—Venogram of two 0.5-cm. polyethylene tubes with vein graft between. Note presence of some collateral venous circulation and normal venous channel diameter in vein graft despite narrowing at the two tubes.

capillaries and small blood vessels. Further demonstration of this external vascularization was given by India ink injection studies. With the vein above and below the prosthesis clamped, India ink injection into the carotid artery on the same side as the prosthesis resulted in marked intimal staining of the vein-graft lining The only way the ink could have reached the intima was through blood vessels in the tissue passing from outside the tube to the vein graft inside. No ink staining of the intima of vein grafts lining solid prostheses was observed. A further demonstration of the importance of the external vascularization through the perforations was given by the frequent finding

Fig. 6.—Venogram of a 2-cm. solid polyethylene vein-lined prosthesis in situ for 15 days. Note abundant venous collateral circulation. Marked thickening and fibrosis of vein graft in tube was found at necropsy.



of maceration and discoloration of the central portion of the vein graft in the solid vein-lined tubes.

Serial venograms were somewhat deceptive in the case of external jugular prostheses, for often the venogram would indicate much greater narrowing of the vein than was found at necropsy. However, the absence of collateral circulation usually could be correlated with an adequately functioning prosthesis, and the presence of many collateral vessels around the tube invariably meant a greatly narrowed or thrombosed tube. These studies demonstrate the frequently observed fact that a blood vessel may be considerably narrowed, with little diminution of flow through it (Figs. 4, 5, and 6).

*Group 3.*—Venous System: Venous homograft lining for prostheses. In this group, there were five homograft-lined perforated polyethylene tubes tied in the superior vena cava. Tube sizes were 410 and 430 (footnote, Table 1). Serial venograms revealed progressive and rapid diminution of the vascular channel through the grafted area, and at necropsy all prostheses were thrombosed, with no blood flow through them. Follow-up times before killing ranged from 11 to 26 days, and venograms as early as 2 days after prosthesis insertion demonstrated narrowing changes when compared with venograms done immediately postoperatively. At necropsy there was marked fibrosis and thickening of all vein grafts, and in four of the five specimens discoloration of the intima was noted. Early function of the homograft-lined prostheses was demonstrated by lack of cyanosis or dyspnea in these animals. That abundant collateral circulation developed in these animals as the prostheses became occluded was shown both by venograms and at dissection before necropsy.

## COMMENT

The results of this study indicate that polyethylene may be added to the list of materials acceptable for use in the nonsuture method of arterial and venous anastomosis. Polyethylene is both inexpensive and easily worked, advantages not possessed by most other prosthetic materials. The only disadvantage of polyethylene tubing as extruded commercially is that the thickness of the wall markedly diminishes the lumen of the vessel into which it is inserted. Therefore, nonsuture anastomoses using such polyethylene tubing put the procedure to a very stringent test, and continued patency testifies to the minor reaction induced by polyethylene in the tissues.[18]

A careful review of the various reports concerning nonsuture anastomosis as applied to arteries in World War II ‡ reveals that in most theaters a variety of prosthetic materials were used and often by surgeons performing their first nonsuture anastomosis. Furthermore, many authors state that the nonsuture method was reserved for the most difficult cases. It is not hard, therefore, to understand the poor results found and to appreciate current pessimism regarding this method of blood vessel anastomosis as applied to arteries. It is by no means clear, however, that this rejection of nonsuture anastomosis is justified. The fundamental objection to nonsuture anastomosis is that there is necessarily narrowing of the vessel into which the prosthesis is tied, and, all other things being equal, this should favor the suture method of anastomosis. However, nonsuture anastomoses are performed more rapidly and give a greater incidence of patency than suture anastomoses in infected wounds, as Blakemore has shown.[8] Our work further justified Blakemore's assertion that the nonsuture method of anastomosis may be effectively used in both small and large arteries.

That external vascularization to autografts is desirable is shown by the much higher incidence of patency with perforated instead of solid polyethylene vein-lined tubing in the venous system. Bencini and Bellinazzo [24] have demonstrated the importance of external vascularization to arterial homografts by wrapping the grafts with polyethylene and finding that the outer layers of the

‡ References 19 to 23.

graft become necrotic. It must be reemphasized that blood-vessel grafting is best done in vascular areas where the adventitia of the graft can receive an external blood supply.[25]

Much better results were obtained with perforated polyethylene vein-lined prostheses inserted in the external jugular vein than were obtained by insertion in a similar manner in the portal system. Although 100% of dogs die after ligation of the portal or superior mesenteric vein and only 1 of 13 dogs in our series died with portal vein prostheses, approximately 50% of the portal vein grafts were thrombosed at necropsy and most of the patent prostheses had thickened vein grafts. We must conclude that there is a progressive fibrosis and thickening of the vein grafts in the polyethylene tubes in the portal circuit, in contrast to a much smaller degree of the same process in the external jugular veins.

It is hard, on the basis of these studies, to recommend the nonsuture method of anastomosis for use in the portal circuit. However, the high incidence of patency and the apparent lack of tendency toward fibrosis in the vein grafts of prostheses inserted in the external jugular vein make it desirable to further assess the method in this area. Bricker and McAfee [26] suggest that bilateral internal jugular vein resection in the older age group may be dangerous and report the case of a 60-year-old man who had undergone such resection and had had the resected venous segment replaced with a femoral vein graft. The nonsuture method of anastomosis would offer the advantages of speed, simplicity, and rigid external support for the vein graft in such cases.

Venous homografts were shown to be undesirable as vein-graft linings for polyethylene prostheses inserted in the superior vena cava. There was inevitable thickening of the vein graft and progression to thrombosis in all cases. However, the results of this study strengthen the impression that nonsuture anastomoses are quite satisfactory in the arterial and peripheral venous systems, at least on a short-term basis. As a technique for restoring vascular continuity, in either simple anastomosis or grafting, nonsuture anastomosis should be considered whenever speed is essential or external support desirable.

SUMMARY

Satisfactory results in both arteries and veins may be achieved with the nonsuture method of anastomosis using perforated polyethylene tubing lined with venous autografts. The superiority of perforated over solid polyethylene vein-lined prostheses in veins has been demonstrated to be due to external vascularization through the perforations to the vein grafts of the perforated tubes. Venous autografts are preferable to venous homografts for lining prostheses in nonsuture anastomoses because of the greater tendency toward thickening and thrombosis in the homograft-lined prostheses. There is a higher incidence of patency and less tendency toward thickening and fibrosis of the vein grafts of prostheses inserted in the external jugular than those inserted into the portal veins.

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# Cardiac Sphincter Substitution by Interpositioned Jejunum

## The Role of the Vagus in Dogs

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Reflux esophagitis is a common complication of certain conditions in which the esophagogastric sphincter fails to function normally or is either incised or excised surgically. This is a frequent clinical observation and has been demonstrated experimentally as well. Recent studies in dogs in this laboratory have shown that excision of the cardioesophageal junction, even when accompanied by a vagotomy and a theoretical 100% resection of parietal cells in the proximal stomach, resulted in a high incidence of erosion and ulceration of the esophagus in the area proximal to the esophagogastrostomy. This complication occurred in all dogs receiving prolonged histamine stimulation and also occurred even when gastroduodenal ulcers failed to develop.*

Further experimental work has shown that when the esophagogastric sphincter is sacrificed, a jejunal segment interposed between the esophagus and intact stomach, accompanied by a bilateral vagotomy and Finney pyloroplasty, protects the esophagus against acid-pepsin digestion.[3] In this new position the jejunal segment itself exhibits remarkable resistance to digestion. It is believed that the interposed jejunal segment behaves as a physiological sphincter. Consistent experimental results have been so encouraging that this operative procedure has been utilized in patients with anatomic and physiologic abnormalities at the cardiac sphincter.[4]

In certain clinical conditions, e. g., reflux esophagitis, it is difficult at times to separate the vagi from the esophagus and a vagotomy is necessary; consequently, a vagotomy was incorporated in our first experimental preparation, accompanied by a drainage procedure. As the clinical indications for cardiac sphincter substitution by an interposed jejunal segment have been extended to conditions other than reflux esophagitis, it has become apparent that a vagotomy may not always be a necessity. Therefore, the question arises whether a vagotomy and drainage procedure are imperative in order to provide adequate protection of the interposed jejunal segment. If with intact vagi the jejunum fares no worse than the stomach and duodenum in similar circumstances, one then may consider omitting the vagotomy and ancillary drainage procedure. Consequently, the operative time and potential morbidity of the procedure should be reduced.

The following experiments were undertaken in an attempt to clarify these questions regarding the role of the vagus not only on the interposed jejunal segment but also on the stomach and duodenum.

### METHOD OF STUDY

Twenty adult mongrel dogs averaging 12.4 kg. in weight were subjected to resections of the distal

Submitted for publication April 18, 1955.

Supported in part by Public Health Grant No. C-2186 and by Initiative 171 Funds from the State of Washington for Research in Biology and Medicine.

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* References 1 and 2.

2 cm. of the esophagus and sufficient cardia of the stomach to insure excision of the cardiac or esophagogastric sphincter. The restoration of gastrointestinal continuity was effected between the esophagus and intact stomach by means of a jejunal segment.

*Operative Procedure.*—All dogs were anesthetized with pentobarbital sodium (Nembutal Sodium) intravenously and supplemented with oxygen and ether as needed, through an endotracheal tube and Palmer respirator. A left subcostal skin incision was used in all cases. The pleuroperitoneal attachment of the hiatal crura to the esophagus was severed, and both vagi were dissected from the esophagus and retracted. The distal 2 cm. of the esophagus and the cardiac sphincter of the stomach were resected. After the cardia was closed, a segment of jejunum then was chosen for transplantation. This jejunal segment was developed usually about 30 to 40 cm. beyond the ligament of Treitz, and the segment varied in length from 6 to 22 cm. An open jejunojejunostomy was done in two layers to restore intestinal continuity. The jejunal segment, with its vascular pedicle, was passed through the transverse mesocolon into the lesser peritoneal sac. The proximal end of the segment then was sutured to the esophagus end-to-end. Care was taken that the segment was isoperistaltic and its mesentery untwisted. An end-to-side anastomosis then was done, approximating the distal end of the segment to a transverse stab wound on the anterior wall of the fundus of the stomach. The rent in the mesocolon was closed loosely about the pedicle. The esophageal hiatus was closed carefully above the esophagojejunostomy.

*Postoperative Care.*—Clyses of 5% dextrose in saline were given for the first three days postoperatively. These animals were allowed water on the fourth day and a puréed diet of milk and Friskies on the seventh day. Most of them regurgitated some food for five to six days and then generally had no difficulty with a regular diet.

*Histamine Stimulation.*—Four dogs were killed during the third and fourth weeks; the remaining sixteen dogs were rested for 45 days postoperatively, then given a 45-day course of daily injections of 30 mg. histamine in yellow wax (beeswax). All were autopsied immediately after death or were killed when they were found to be moribund. Those animals surviving the course of histamine administration were killed. Microscopic examination was made of the esophagojejunal and jejunogastric anastomoses and any other areas where indicated.

## RESULTS AND COMMENT

Table 1 contains an analysis of the results obtained. There were no lesions of the esophagus in any animal. Concerning jejunal vulnerability, 9 of the 20 had some degree of ulceration, including 4 instances of perforation; however, in 7 of these 9 animals there was ulceration of the stomach and/or duodenum of equal or greater severity. In three of the four dogs with jejunal segment perforations there were widespread perforative ulcerations in the stomach and duodenum. The two dogs which revealed jejunal ulceration severer than those in the stomach or duodenum merit some description. One of the animals (Y-197) had the shortest segment (6 cm.) of the series. This segment was totally immobilized in dense fibrous adhesions, probably the result of an anastomotic leak. This would tend to reduce the effective peristaltic action of the segment, which is an important protective mechanism not only for the esophagus but also for the jejunal segment. In the other animal (Y-215) there was a diffuse inflammation and superficial ulceration of the distal one-half of the jejunal segment as well as of the duodenum. This dog was observed to be vomiting food and blood after one injection of 30 mg. of histamine in yellow wax and was killed because it was assumed it was suffering from early severe ulcerations.

Four dogs were killed during the third and fourth weeks postoperatively because of severe vomiting. None revealed either esophageal or jejunal pathology. An explanation for the vomiting was possible in three of these animals. Two animals were suffering from lower small-bowel obstruction. One received histamine prematurely and exhibited an acute gastric ulcer. No explanation was possible in the fourth animal.

All dogs receiving histamine showed some degree of gastroduodenal ulceration; in fact, 75% of them died of perforations in either the stomach or duodenum.

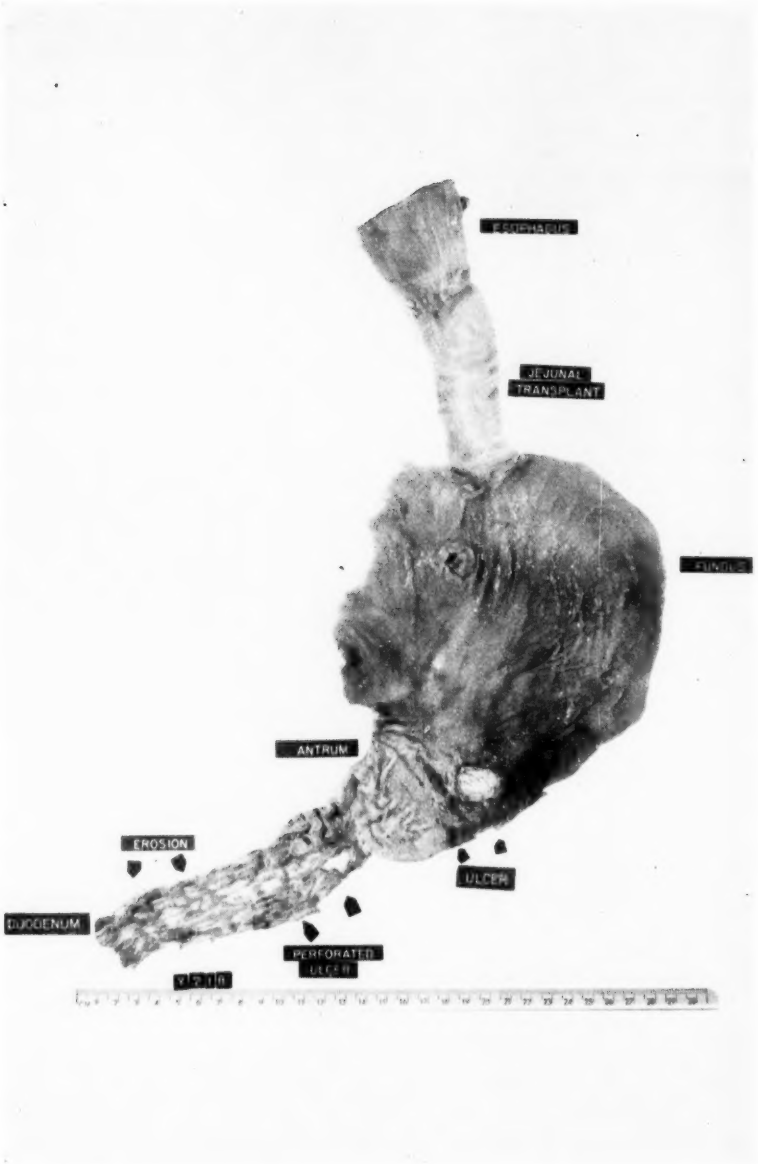
Since none of the dogs showed esophageal disease, it can be assumed that the length of the segments was adequate. Jejunal disease was always limited to the distal 5 cm. of the segment, and none of the segments were shorter than 6 cm. This is similar to the results of other experiments on this

TABLE 1.—*Operation: Excision of Cardiac Sphincter, Interposition of an Isoperistaltic Segment of Jejunum Break Between the Esophagus and Stomach; Intact Vagi and Pylorus*

| Dog | Total Survival, Days | Days of Histamine | Length of Jejunal Segment, Cm. | Origin of Distal End of the Jejunal Segment from the Pylorus (Site of Jejunogastrostomy), Cm | Gross and Microscopic Appearance of Tissues; Degree of Ulceration† | | | | Comment |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | Esophagus | Jejunum | Stomach | Duodenum | |
| Y-186 | 54 | 7 | 18 | 204 | 0 | 0 | 4 | 2, 3 | Died of gastric perforation |
| Y-189 | 76 | 31 | 13 | 89 | 0 | 1 | 3 | 3, 4 | Died of duodenal perforation |
| Y-191 | 92 | 46 | 18 | 118 | 0 | 0 | 0 | 1 | Killed after 45 days histamine |
| Y-190 | 48 | 3 | 14 | 110 | 0 | 0 | 4 | 1 | Died of gastric perforation |
| Y-193 | 23 | 0 | 11 | 117 | 0 | 0 | 2 | 0 | Killed, refused to eat |
| Y-194 | 63 | 18 | 16 | 100 | 0 | 0 | 0 | 3, 4 | Died of duodenal perforation |
| Y-197* | 52 | 7 | 6 | 102 | 0 | 4 | 1 | 3 | Died of jejunal segment perforation |
| Y-200 | 49 | 4 | 12 | 114 | 0 | 0 | 1 | 3, 4 | Died of duodenal perforation |
| Y-201 | 49 | 4 | 11 | 147 | 0 | 4 | 4 | 3, 4 | Died of multiple perforation |
| Y-203 | 51 | 6 | 12 | 122 | 0 | 0 | 4 | 2 | Died of gastric perforation |
| Y-204 | 64 | 19 | 10 | 106 | 0 | 2 | 2 | 3 | Killed; tarry stools |
| Y-205 | 48 | 3 | 22 | 102 | 0 | 4 | 4 | 4 | Died of multiple perforation |
| Y-206 | 27 | 2 | 14 | 140 | 0 | 0 | 2 | 2 | Histamine given by mistake; killed; vomiting |
| Y-208 | 19 | 0 | 16 | 144 | 0 | 0 | 0 | 0 | Died of strangulated ileum |
| Y-210 | 27 | 0 | 7 | 112 | 0 | 0 | 0 | 0 | Died of strangulated ileum |
| Y-212 | 50 | 5 | 6 | 71 | 0 | 4 | 2 | 1, 3, 4 | Died of duodenal and jejunal perforation |
| Y-214 | 47 | 2 | 7 | 95 | 0 | 1 | 4 | 1, 3 | Died of gastric perforation |
| Y-215* | 46 | 1 | 15 | 109 | 0 | 1, 2 | 0 | 1 | Killed; vomiting blood |
| Y-217 | 51 | 6 | 12 | 78 | 0 | 1, 2 | 0 | 3, 4 | Died of duodenal perforation |
| Y-218 | 47 | 2 | 10 | 118 | 0 | 0 | 2 | 3, 4 | Died of duodenal perforation |

* Only two dogs that had jejunal ulceration severer than elsewhere.

† 1—Reddening or inflammation
2—Mucosa ulcerated to submucosa (erosion)
3—Ulcer through submucosa into muscularis
4—Perforation of wall




Dog Y-218. This autopsy specimen exhibits the effects of two days of histamine stimulation. This 10-cm. jejunal segment and esophagus are normal, while there are deep ulcers of the stomach and duodenum.

problem. The average length of the segments in this series was 12 cm. This length was dictated only by the anatomy of the particular vascular pattern found in the jejunum of each animal. There was no correlation between either the incidence of jejunal ulceration and the segment length or the intestinal site from which the segment was obtained. The segments were removed an average distance of 110 cm. below the pylorus, as close as 71 cm. and as distant as 204 cm. The most distal portion of the segment would, of course, be utilized in the jejunogastric anastomosis and, therefore, would be adjacent to the gastric chyme. This tends to support the thesis that no important inherent sensitivity of the small bowel to acid-peptic ulceration exists.† Since all dogs undergoing histamine stimulation showed gastroduodenal ulcerations and only nine, or 45%, showed concomitant, though generally less severe, jejunal disease and only two, or 10%, showed severer jejunal involvement than elsewhere, one may deduce that the sensitivity and susceptibility of the interposed segment to acid-peptic digestion is less in its new position than the duodenum or even the stomach. The free peristaltic action of the segment and the alkalinizing salivary juices undoubtedly contribute to its protection.

In comparing this series with previous work from this laboratory,[3] Table 2 gives some insight into the major differences and similarities. In neither series was there evidence of esophageal pathology; consequently, the vagus nerves have no influence in esophageal protection. Therefore, one may conclude that the interposed jejunal segment alone protects the esophagus against the acid-peptic digestion which consistently occurred when the cardiac sphincter was sacrificed and a direct esophagogastric anastomosis made.[2] Consequently, the major interest centers about comparing the effect of vagotomy, with or without a drainage procedure, with the effect of intact vagi on the jejunal segment, stomach, and duodenum.

It is apparent that the effect on the jejunal segment is comparable in animals with that of vagotomy and intact pylorus, vagotomy and Ramstedt pyloromyotomy, and, in this series of animals, with intact vagi and pylorus. Only when an adequate drainage procedure, e. g., Finney pyloroplasty, is added to the vagotomized animal is almost complete protection accorded the jejunal segment, even in the presence of histamine stimulation.

In terms of the effect of vagotomy versus intact vagi in this experimental preparation on gastric and duodenal pathology, it is obvious that, under histamine stimulation, those animals with intact vagi fare worse than vagotomized animals with or without adjunct drainage procedures. Also, it appears that a Finney pyloroplasty gives added protection to the stomach and duodenum as

TABLE 2.—*A Comparison of the Effect of the Vagus in the Interposed Jejunal Segment Operation*

| | Esophagitis | Jejunum | Stomach | Duodenum |
|---|---|---|---|---|
| Previous series* | | | | |
| Standard operation† plus | | | | |
| Vagotomy with intact pylorus..... | 0% | 40% Ulcers<br>20% Perforations | 60% Ulcers<br>No perforations | 80% Ulcers<br>50% Perforations |
| Vagotomy with Ramstedt pyloromyotomy | 0% | 50% Ulcers<br>10% Perforations | 40% Ulcers<br>20% Perforations | 90% Ulcers<br>30% Perforations |
| Vagotomy with Finney pyloroplasty..... | 0% | 10% Ulcers<br>No perforations | 50% Ulcers<br>20% Perforations | 40% Ulcers<br>20% Perforations |
| Present series | | | | |
| Standard operation with intact vagi..... | 0% | 45% Ulcers<br>10% Perforations | 87% Ulcers<br>37% Perforations | 100% Ulcers<br>50% Perforations |

* Reference 4.

† The standard operation consists of excision of the distal 2 to 3 cm. of esophagus and cardia sufficient only to insure excision of the esophagogastric sphincter. A jejunal segment of variable length, distal to the ligament of Treitz, is interposed between the esophagus and the intact stomach in order to restore continuity. Intestinal continuity at the original site of the jejunal segment is made by jejunojejunostomy. All dogs were stimulated by 30 mg. of histamine in yellow wax daily.

† References 5, 6, and 7.

well as the jejunal segment. This observation tends to substantiate the contention of Ferguson [8] that the parietal cell secretion, in response to histamine stimulation, is decreased when a vagotomy is performed.

While jejunal ulceration in this present series with intact vagi is high and essentially in agreement with the work of others,[9] the incidence and severity of ulcerative disease of the stomach and duodenum far exceeds that which occurred in the jejunal segment. Consequently, it appears that even in the presence of intact vagi the interposed jejunal segment maintains a greater resistance to acid-peptic ulceration and perforation than either the stomach or the duodenum.

## SUMMARY AND CONCLUSIONS

1. A vagotomy exerts no influence in the prevention of esophagitis when a jejunal segment is interposed between the esophagus and the stomach.

2. The interposed jejunal segment protects the esophagus against the development of esophagitis in the presence of intact or divided vagi.

3. Vagotomy, with or without an ancillary drainage procedure, however, results in a decrease in the incidence and severity of gastric and duodenal ulcerations even in the histamine-stimulated animal.

4. With intact vagi plus histamine stimulation the interposed jejunal segment exhibits greater resistance to acid-peptic digestion than either the stomach or the duodenum.

5. Vagotomy does not appear to be an essential part of the interposition operation, although clinically it may be indicated by paraesophageal fibrosis or by the presence of ulcerative disease other than in the esophagus itself.

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# Hemophilic Arthropathy

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The hemarthropathy clinic at The Jefferson Medical College Hospital, organized and conducted by the combined direction of the departments of hematology and orthopaedic surgery, provides adequate material for the study of hemophilic arthropathy. Up to the present time 117 hemophiliacs are available for study. In this presentation, the roentgenological features of hemophilic arthropathy are reviewed, the gross and microscopic alterations are noted, the pathogenesis of the disorder is reconstructed, a new method of grading the severity of the arthropathy is presented, and, finally, the correction of deformities by surgical intervention is discussed.

## ROENTGENOLOGIC FEATURES

The roentgenologic features of this disorder have been recorded by many observers. The most significant findings are the following: (1) There is increased density in the synovial and capsular tissues; (2) cystic areas are noted in the subchondral and cancellous bone; (3) in advanced cases the bone ends are broader than normal and the joint space may be narrowed; (4) collapse of the bony trabeculae may occur, producing an angular deformity; (5) spurring of the tips of the olecranon, coronoid process of the ulna, the poles of the patella, and the tibial spines are common findings; (6) there is broadening and deepening of the intercondylar notch of the femur, and (7) pronounced subchondral sclerosis occurs in the advanced stages of the arthritis.

Some cases exhibited rapid increase in the size of the osteogenic growth center. This is noted frequently in the epiphyses of the elbow joint, particularly the radial head. Overgrowth of the epiphyses in all their dimensions is a common finding; this is responsible for the characteristic knobbiness so frequently exhibited by hemophilic joints. Occasionally, premature closure of a portion or the entire epiphyseal plate may occur.

## PATHOLOGY

We have been fortunate in obtaining several hemophilic joints (two knee joints and two shoulder joints) from a hemophiliac, 27 years of age, who came to autopsy. In addition, tissues obtained from several joints which were exposed during surgical procedures were also studied and the observations recorded.

*Macroscopic Findings.*—Right Shoulder: The entire articular surface is stained a chocolate color, and its periphery is irregular in outline. No vestige of hyaline cartilage remains. The subchondral bone is eburnated and peppered with numerous crater-like defects that vary in size and shape.

The stained synovial membrance is thin and exhibits fraying and some synovial tabs. The entire musculotendinous cuff shows advanced degenerative changes, comprising shredding, lamination, and fibrous tissue proliferation.

Left Shoulder Joint: In contrast to the profound alteration observed in the right shoulder, gross examination of the left shoulder fails to show any abnormalities.

Right Knee Joint: The cartilage is severely pitted and also exhibits clefts and fissures of varying size and depth from which villi project. Soft tissue alterations in this

Recorded for publication Sept. 16, 1955.

Read before the Section on Orthopedic Surgery at the 104th Annual Meeting of the American Medical Association, Atlantic City, N. J., June 9, 1955.

joint are pronounced. The synovial membrane and subsynovial tissues are markedly thickened. Numerous fine villi varying in length cover the entire synovial lining and practically obliterate the suprapatellar pouch.

Left Knee Joint: The alterations noted in this joint are essentially similar to those observed in the right knee.

*Microscopic Features.*—Articular Cartilage: This structure exhibits marked fibrillation of its collagen fibers, fragmentation, and thinning. In some areas, the entire thickness of the cartilage exhibits complete disintegration. In other areas, the presence of Weichselbaum's lacunae and the fragmentation of the cartilage overlying the lacunae are clearly discernible. In several areas the cartilage is replaced by vascular proliferating granulation tissue extending from the marrow spaces.

Subchondral and Cancellous Bone: Marked irregular thinning of the subchondral bone is noted. Many marrow spaces adjacent to the subchondral bone contain an abundance of vascular proliferating connective tissue which appears to cause absorption of the surrounding bony trabeculae.

Synovial Membrane: The synovial membrane overlying the fat pad is markedly thickened, hyperplastic, and endowed richly with vascular channels. Villi varying in size and shape are discernible. Most of the synovial cells contain fine granules or iron pigment; in some, large deposits of the pigment are observed.

The subsynovial layer is exceedingly vascular and scattered throughout its entire thickness are large clumps of pigment which either is lying free or is in macrophages.

*Pathogenesis of Arthropathy in the Hemophiliac.*—The aforementioned clinical, roentgenographic, and microscopic observations shed some light on the pathogenesis of this disorder. As far as one can determine from a clinical viewpoint, a hemophiliac is born with normal joints. However, the first hemorrhage into a joint may occur very early in childhood—the youngest patient with hemarthrosis in this series was 12 months old—however, most occur after the age of four years.

Repeated insults to the joint by numerous and frequent hemorrhages produce tissue changes which are irreversible. The essential response of these tissues is a hyperplastic process producing a villous synovitis and thickening of the subsynovial tissues by marked fibrous tissue proliferation. These tissues are laden with blood pigment.

*Cyst Formation.*—The cystic areas and clefts noted in the articular cartilage may arise from coalescence of Weichselbaum's lacunae, by replacement of deteriorated areas of articular cartilage, by connective tissue, or by the destruction of the overlying subchondral bone and cartilage by vascular connective tissue originating in the marrow spaces immediately adjacent to the subchondral bone. All these modes of origin are noted in the microscopic study recorded herein.

*Classification of Hemophilic Arthritis.*—In this study an attempt has been made to classify hemophilic arthropathy into four grades which correlate the clinical manifestations, the pathologic alterations, and the observations noted roentgenologically.

Grade I: This is the earliest form of arthritis encountered. Essentially, the changes are limited to soft-tissue elements. Clinically, the joint exhibits no restriction in its arcs of motion and no deformities are discernible. Slight atrophy of the muscles about the joint may be present. Some thickening of the synovial and subsynovial tissues is discernible. Roentgenograms exhibit no bony abnormalities except for some slight generalized decreased density of the bone ends.

Grade II: Deformities of joints in one plane are discernible. The deformities are due entirely to contractures implicating the capsular and pericapsular tissues, resulting in a loss of a few degrees of the normal arcs of motion. The bone ends exhibit slight broadening and the first indications, in a mild form, of knobbiness of the joint are manifest. Roentgenologic study reveals no incongruity of the joint surfaces, and the joint space is well preserved.

Grade III: Joints in this stage of development of the arthritic process show fixed deformities which in some incidents are in more than one plane. Examples of this grade are readily demonstrable in the knee, elbow, and subtalar joints. The knee may exhibit flexion, genu valgum, and external rotation of the tibia on the femur. In addition, clinical examination reveals marked atrophy of the muscles motorizing the joint, pronounced pericapsular and capsular thickening, and knobbiness of the bone ends. Roentgenologically, one notes irregular overgrowth of the epiphysis and varying degrees of incongruity of the articular surfaces. Clefts and punch-out areas in the cartilage and cysts in the subchondral and cancellous bone are not yet prominent features and may be absent.

Grade IV: This stage embraces the severest alterations encountered in this study. Existing deformities are fixed and pronounced. Marked knobbiness is a constant feature, and in some instances fibrous ankylosis exists. Fibrous ankylosis is a frequent finding in the patellofemoral joint. Roentgenologic study reveals bony sclerosis to be the outstanding feature. In addition, part or all of the joint space is thinned or obliterated completely.

MANAGEMENT

*Massive Hemarthrosis.* — Patients with acute massive hemarthroses are admitted to the hospital and put at complete bed rest. The blood defect is immediately studied and corrected by the administration of whole blood or plasma.

The joint is put at complete rest and surrounded by ice packs. Where possible, an elastic compression bandage is applied. Wherever feasible, aspiration of blood is performed routinely in this clinic and 1 cc. of hydrocortisone is injected into the joint. In fact, if necessary, repeated aspirations are done. This is followed by the application of a compression bandage. As yet, no unfavorable sequelae have followed joint aspiration. We are of the opinion that spreading factors, such as hyaluronidase, should not be employed in massive intra-articular hemorrhage. This is based on the clinical observation that joints treated by such spreading agents return to normalcy much more slowly than joints treated by aspiration and compression. Hyaluronidase favors rapid absorption of intra-articular blood, particularly when under great tension. However, when this occurs the synovial membrane is forced to carry a tremendous work load within a brief period of time and is subjected suddenly to a concentration of noxious factors in the blood. It becomes apparent that repeated insults of this nature have serious deleterious effects on the joint tissues and will enhance development of arthritic alterations.

Active motion within the painless arcs is instituted early; however, no weight-bearing is permitted at this time. If the knee joint is implicated, care must be taken to prevent a flexion deformity of the joint. Traction may be applied after the blood is aspirated and bleeding is controlled.

*Correction of Joint Deformities.* — After acute hemarthroses, knee joints, especially in children, show a great tendency to develop fixed flexion deformities. If after the measures previously described complete correction is not achieved, a long leg brace with a circular spring at the knee joint is worn for part of the day and during the sleeping hours. The spring is so designed that it exerts continuous extension at the knee joint and yet allows some voluntary flexion. While wearing the brace, the patient performs resistance exercises on a regulated regimen. The brace is discarded just as soon as complete extension can be performed voluntarily and the tendency toward development of a flexion deformity is overcome. Mild flexion deformities which fail to respond to this method can be corrected by continuous skin traction and wedging plaster casts. Moderate and severe flexion deformities are usually associated with valgus at the knee joint and external rotation of the tibia on the femur. Regardless of the methods employed, we have never been able to correct the last two components of the deformity. Furthermore, the flexion deformity in these joints

may be corrected, but invariably the tibia assumes a position of posterior subluxation. Such hopeless cases have been the stimulus for investigating a surgical approach to the problem. The hazards associated with surgery on the hemophiliac are well known; nevertheless, we believe that in properly selected cases and with adequate pre- and postoperative management surgical correction of some of these deformities is possible. In the selection of cases, patients with severe blood defects are excluded; only patients with mild or moderate defects of Grades I and II are considered suitable subjects. By observing these rules in this clinic, the following surgical procedures have been performed without any unfavorable sequelae: one arthrodesis of the knee joint, one arthrodesis of the hip joint, one massive excision of necrotic tissue and replacement with skin grafts, two shelf operations, and two derotation osteotomies for congenital dislocation of the hip joints.

In conclusion, I wish to state emphatically that surgery in the hemophiliac is dangerous and should never be attempted except in those cases in which all other means have been exhausted, and then only if the patient's blood is of Grade I and II and if the personnel and physical equipment are available to administer adequate pre- and postoperative management.

# Diagnostic Accuracy of Hepatic Metastases at Laparotomy

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One of the most important steps in the procedures of the surgeon on opening the abdomen of a patient with cancer is to inspect the liver with care, in order to determine possibly the presence or absence of metastatic cancer. The determination is one which may change completely the nature and degree of the contemplated operation. For example, some surgeons rigidly refuse to proceed with a gastrectomy, pancreatectomy, or rectal resection in the presence of hepatic metastases, regardless of their small size or small number, whereas other surgeons with different points of view would proceed with the resection and, even under favorable circumstances, practice metastasectomy. Moreover, an operation applied for obvious palliation is often of more limited scope than the radical procedures designed with hope for cure.

We thought it to be of interest to determine the accuracy of our diagnosis of freedom or noninvolvement of the liver by metastatic cancer at the time of laparotomy for cancer of the gastrointestinal tract. Over a 25-year period of surgical effort we selected a group of 100 consecutive patients whose operative experience fulfilled these criteria: (*a*) The operations were for cancer of the gastrointestinal tract; (*b*) a laparotomy or laparothoracotomy was done in each instance; (*c*) the livers were carefully inspected by us and found to be free of detectable evidence of metastatic cancer; (*d*) the 100 patients died in the hospital within 1 to 30 postoperative days, and (*e*) postmortem examinations were made in all cases. The data so secured furnished information enabling us to judge the accuracy of our clinical appreciation of the normalcy of the liver. We have no instances in which we erroneously thought metastases to be present in the liver.

In explanation it should be stated that these operative deaths were largely not in patients who had undergone radical surgical procedures, although some were in this category; instead they were the natural although premature consequence of abdominal exploration for cancers deemed nonresectable for other reasons than hepatic metastasis, i. e., peritoneal carcinosis, retroperitoneal and mesenteric involvement, etc. At the Memorial Hospital, exploratory laparotomy, for example, in some years has had a higher

TABLE 1.—*Diagnostic Accuracy of Hepatic Metastases at Laparotomy in One Hundred Cases Wherein the Liver was Declared to be Normal*

| Site of Primary Cancer | Postmortem State of Liver: Normal | Postmortem State of Liver: Metastases Present | Total Cases |
|---|---|---|---|
| Esophagus | 9 | 1 | 10 |
| Stomach | 34 | 3 | 37 |
| Duodenum | 1 | 0 | 1 |
| Pancreas | 6 | 1 | 7 |
| Cecum | 3 | 0 | 3 |
| Hepatic flexure colon | 2 | 0 | 2 |
| Splenic flexure colon | 1 | 0 | 1 |
| Descending colon | 1 | 0 | 1 |
| Sigmoid colon | 10 | 0 | 10 |
| Rectosigmoid | 1 | 0 | 1 |
| Rectum | 27 | 0 | 27 |
| | 95 | 5 | 100 |

Conclusion: Incorrect diagnosis of state of liver at laparotomy, 5%; diagnostic accuracy by surgeon on inspection of liver, 95%.

Submitted for publication June 1, 1955.
From the Memorial Center.

operative mortality than gastrectomy. Such patients with the handicap of the persistent cancer plus the ardors of the operative experience do not receive that daily increment of improvement during their convalescence that is necessary for recovery.

It has been the experience of many a surgeon to resect a gastrointestinal cancer with intent and hope for cure only shortly to palpate a nodular hard liver, indicative of hepatic metastases. At such a time he is assailed by doubt as to the thoroughness of his inspection of the liver and questions his ability to detect small hepatic metastases when present. However, it is not necessarily true that cryptic latent metastases may be present in the liver with considerable frequency, only to reappear disconcertingly at a later day; it is equally possible that these patients have residual cancer left at the operative site, within the viscera or small adjacent nodes, which may form the nidus for subsequent migration to the liver. Some credence to the latter hypothesis may be found in the results of this study.

Postmortem examination of the 100 livers thought to be normal by the surgeon at laparotomy revealed unsuspected metastatic cancer in 5. Therefore, it may be said either way: that our incorrect diagnosis of the freedom of the liver from metastasis was 5%, or that our clinical diagnostic accuracy was 95%. (Table 1.) A 5% chance of undetectable cancer being present in the liver should never deter the surgeon from performing the indicated resection. On the other hand, when we study the individual cases in which the errors were made, it is noted that in three of these five the pathologist himself on gross inspection and sectioning of the livers was not cognizant of the presence of cancer; only subsequent microscopical study of the liver revealed the unsuspected presence of diffuse metastatic cancer. The correctable or inexcusable error of the surgeon, therefore, was only 2%.

TABLE 2.—*Incorrect Diagnosis of Hepatic Metastases by Surgeon at Time of Laparotomy*

| Site of Primary Cancer | Sex | Age | Cause of Death | Post-operative Day of Death | Site of Metastases | Diagnosis of Metastases at Operation | Gross Diagnosis of Pathologist at Necropsy | Microscopical Pathological Diagnosis | Comments |
|---|---|---|---|---|---|---|---|---|---|
| 1. Esophagus | M | 62 | Coronary thrombosis | 1 | 1 cm. deposit near right hepatic vein | Negative | Intrahepatic 1 cm. nodule near right hepatic vein | Metastatic epidermoid carcinoma | Difficult region to detect |
| 2. Stomach | M | 73 | Coronary thrombosis | 14 | Diffuse hepatic metastases (microscopic) | Negative | No suspicion of cancer in liver | Diffuse invasive cancer | Microscopic diagnosis only |
| 3. Stomach | M | 69 | Hepatic insufficiency | 8 | Diffuse hepatic metastases (microscopic) | Negative | No suspicion of cancer in liver | Diffusely metastatic adenocarcinoma | Microscopic diagnosis only |
| 4. Stomach | M | 65 | Atelectasis Pneumonia | 4 | 1.5 cm. deposit in quadrate lobe | Negative | Solitary metastasis quadrate lobe | Metastatic adenocarcinoma | Definite clinical error in diagnosis |
| 5. Pancreas | M | 71 | Renal failure Jaundice Cholangitis | 1 | Diffuse hepatic metastases (microscopic) | Negative | No suspicion of cancer in liver | Metastatic mucinous adenocarcinoma | Microscopic diagnosis only |

Comment: In a series of 100 laparotomies and laparothoracotomies for cancers of the gastrointestinal tract, at which time we considered the liver to be clinically free of metastatic involvement, subsequent postmortem examination revealed undetected metastases in 5 livers (error—5%). However, an analysis reveals that in 3 of the 5 cases, the pathologist, himself, on gross inspection and sectioning of the livers was not cognizant of the presence of cancer. The correctable or inexcusable error of the surgeon, therefore, was only 2%.

# The Hormonal Cause of the So-Called Varicose Veins of Pregnancy

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Many aspects of superficial varicose veins of the lower extremities complicating pregnancy have been controversial.* The present study offers explanations for some of the controversies.

The superficial venous disorders of the lower extremities during pregnancy have universally been identified as the classical superficial saphenous varicosities. We recently differentiated from the well-known saphenous varix a hitherto undescribed, completely different, superficial venous disorder which is limited to the pregnant state.[1] We applied to it the term "angiectid." †

A review of the literature indicated that the failure to differentiate the angiectid from the saphenous varix in the past was the cause for many of the controversies. This shortcoming is unexplainable because the two disorders differ so very much. The angiectid does not resemble the long, cord-like, subcutaneous saphenous varicosity. The angiectid is a small, superficial, intradermal, raised, sharply circumscribed clump of bluish vessels that has a predilection for specific sites (Figure). It often is warm and tender. With the exception of the angiectids, the venous systems of these patients generally are normal.

The pain caused by the angiectid is in contrast to the heaviness and fatigue of the legs occasioned by the saphenous varix. The pain is present in most cases and always is somewhat disabling. Often it is severe enough to prevent the patient from standing for more than five minutes. When pain is present, the angiectid is warm, tender, and tense. These characteristics are confined to the angiectid and do not involve the surrounding tissue. No thrombophlebitis or ulceration has been noted to account for the pain or signs.

Many clinical features of the angiectid implied a relationship between the disorder and changes in the normal balance of estrogen and progerterone. In all instances the women were young and the angiectids appeared initially during the first trimester of one of their first three pregnancies. About half of the patients found that the angiectids disappeared completely by the sixth postpartum week. The remainder had faint intradermal vestiges which in some instances were associated with premenstrual changes and mild pain. The classical saphenous varices, in contrast, were present previous to pregnancy, enlarged during pregnancy, and returned to their pregravid status after delivery.

Submitted for publication March 30, 1955.

This study was aided by a grant from the Squibb Institute for Medical Research.

Presented at the Obstetrical Society of Philadelphia on Oct. 1, 1953.

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* References 1 to 8.

† Derivation of the new term, "angiectid": angi (Gr. *angeion*, a vessel); ect (Gr. *ektasis*, a stretching); id (Gr. *idios*, peculiar: in this instance, peculiar to pregnancy). We have limited this term to the intradermal vessel dilatation in the lower extremities of pregnant women, as described in this paper.

Angiectids on upper calf.

The purpose of the present study was to determine a possible relationship between the angiectid (vascular system changes) and variations in the amounts of the circulating female sex hormones and to offer explanations for some disputed aspects of the so-called varicose veins of the lower extremities complicating pregnancy.

## METHOD OF STUDY

The data in this study were obtained by endocrinologic obstetric, peripheral vascular, and photographic surveys of 41 pregnant patients who were referred to us from the obstetric outpatient clinic of the Jefferson Medical College Hospital for the study of varicose veins of the lower extremities complicating pregnancy.

Four women had the classical superficial saphenous varicosities only. Thirty-seven patients had angiectids. The latter were 10% of the prenatal cases admitted to the obstetric clinic during the course of the study.

The endocrinologic evaluation consisted of medical histories, physical examinations, and laboratory studies of the 41 patients. The histories and physical examinations were within normal limits.[1] The laboratory studies which provided positive findings included titrations of the estrogenic hormone in the serum by Rakoff's modification of the Fluhman method,[9] evaluations of the amount of chorionic gonadotropin in the serum by the use of the rat-ovary hyperemia reaction as the end-point,[10] determinations of urinary pregnanediol levels by the method of Sommerville, Gough, and Marrian,[11] titrations of the urinary 17-ketosteroids, and studies of estrogen clearance.[12]

Other laboratory procedures included the evaluation of blood calcium levels, of the bleeding, clotting, coagulation, and prothrombin indices, and of liver and kidney function studies.

## RESULTS OF THE HORMONE ASSAYS AND OTHER STUDIES

Determinations of the amounts of urinary preganediol, urinary 17-ketosteroids, serum estrogen, and serum chorionic gonadotropin were done by the previously mentioned assay methods in 25 of the 41 patients with saphenous varices or angiectids and for control purposes in 10 uncomplicated pregnancies. In the latter cases the results were normal.

The levels of serum chorionic gonadotropin were determined periodically in the 25 cases and were consistently within the normal range for the period of gestation. The 17-ketosteroids were also within normal limits in every instance.

TABLE 1.—*Serum Estrogen and Urinary Preganediol Levels* with Saphenous Varicosities*

| Weeks of Pregnancy | Estrogen † | Pregnanediol, Mg./24 Hr. |
|---|---|---|
| 22 | 60 | 30.3 |
| 28 | 100 | .... |
| 35 | .. | 45.2 |
| 36 | 100 | .... |

* All estrogen and pregnanediol titers were normal.
† Fluhman mouse units per 100 cc. of blood.

TABLE 2.—*Serum Estrogen and Urinary Pregnanediol Levels *of Patients with Symptomless Angiectids*

| Weeks of Pregnancy | Estrogen † | Pregnanediol, Mg./24 Hr. |
|---|---|---|
| 14 | 20 | .... |
| 16 | 40 | 17.6 |
| 22 | 60 | 21.6 |
| 28 | 60 | .... |
| 30 | 80 | .... |
| 35 | 100 | 51.3 |

* All estrogen and pregnanediol titers were normal.
† Fluhman mouse units per 100 cc. of blood.

TABLE 3.—*Serum Estrogen and Urinary Pregnanediol Levels of Fifteen Patients with Symptomatic Angiectids*

| Weeks of Pregnancy | Estrogen * | Pregnanediol, Mg./24 Hr. |
|---|---|---|
| 12 | <10 | 4.0 |
| 16 | <10 | 6.6 |
| 19 | <10 | 6.4 |
| 20 | 20 | 6.5 |
| | 10 | 10.0 |
| | <20 | .... |
| 26 | <10 | 15.1 |
| | 30 | 20.3 |
| | 30 | 28.2 † |
| | 30 | 6.2 |
| 28 | 20 | 22.7 |
| | 50 | 34.8 † |
| 29 | 70 † | 15.4 |
| | 30 | .... |
| 30 | 60 † | .... |
| | 40 | 4.1 |
| | .... | 10.0 |
| | 50 | 34.8 † |
| | 20 | 20.2 |
| 35 | <20 | 51.3 † |
| 36 | 20 | 27.6 |
| | 40 | 25.0 |
| 38 | 60 | 30.0 |
| 39 | 60 | 25.1 |

* Fluhman mouse units per 100 cc. of blood.
† Normal titer (all others less than normal).

The amounts of estrogen and pregnanediol were normal in the four patients with saphenous varices (Table 1).

Variations in estrogen and/or pregnanediol levels ‡ were noted among the 31 patients with angiectids. The six patients with symptom-free angiectids had consistently low normal titers of the hormones (Table 2). With a single exception,§ the 15 women with painful, disabling angiectids had subnormal amounts of one or both hormones (Table 3). All titers were subnormal in 11 patients, of whom 3 had assays for one hormone and 8 ‖ had assays for both hormones. In each of the remaining three cases the titer of one of the two hormones assayed was subnormal.

There was further evidence of the relationship between the estrogen-progesterone titers and the clinical status of the angiectid. Two women had symptomless angiectids and normal assay levels of the hormones during the initial months of their pregnancies. When the angiectids caused pain and disability at a later date, the levels of both hormones were subnormal.

The degree of pain and disability caused by the angiectid frequently was related to the level of the subnormal estrogen titer (Table 4). The slightly or moderately disabled patients had varying estrogen levels, none of which was markedly diminished. Of the eight severely disabled women, six had markedly diminished estrogen levels. In addition, these six patients had the only angiectids with tension spots,¶ the most disturbing type of angiectid.

There was no correlation between the degrees of pain (and disability) and the pregnanediol levels.

Estrogen clearance studies were done in three cases and were normal. Liver and kidney function results, blood calcium levels, and bleeding, prothrombin, clotting, and coagulation times were within normal limits.

‡ Specimens for estrogen and pregnanediol assays were collected on the same day.

§ Based on a single evaluation for estrogen. The titer was normal.

‖ Five of the eight women had two to four assays of both hormones. All titers were subnormal.

¶ Focal overdistentions with broken surfaces.

TABLE 4.—*Relationship of Hormone Assay Levels to Degrees of Disability Due to Angiectids*

| | Estrogen | | | | Pregnanediol | | | |
|---|---|---|---|---|---|---|---|---|
| | | Diminished | | | | Diminished | | |
| Degree of Disability | Normal | Slight | Moderate | Marked | Normal | Slight | Moderate | Marked |
| Severe | 0 | 0 | 2 | 6 | 2 | 0 | 3 | 3 |
| Slight to moderate | 2 | 2 | 3 | 0 | 0 | 0 | 2 | 1 |

COMMENT

The failure in the past to recognize the angiectid was the cause for many disagreements about the treatment of superficial varicose veins of the lower extremities during pregnancy. The latter had always been accepted as being classical, superficial saphenous varicosities. The presence of both disorders unknowingly led to the formation of the two divergent schools of treatment. The active therapists, mainly consultants, who did not have the opportunity of following the course of the venous changes during pregnancy utilized surgery and/or the injection of sclerosing solutions as for the saphenous varicosities of their nonpregnant patients.# The conservative group, primarily obstetricians, unknowingly based its therapy on the presence of the angiectid. They temporized with supportive stockings and rest,* because they found that the majority of the saphenous varices, as the angiectid, appeared in the early months of pregnancy and disappeared with delivery.† They also found that these varices generally re-formed after treatment with surgery or injections,‡ which paralleled our experiences with the angiectids. This observation may provide an answer for the differing results reported with the active therapies. The effectiveness of the agent in each series depended upon and must have varied inversely with the number of treated angiectids.

Since the conservative temporizing measures have not been satisfactory,[13] it may be added that we found the female sex hormones to be effective therapeutic agents for the angiectids in this small group of 15 women with painful, disabling angiectids. Since the majority (86%) of the patients had an estrogen deficiency, estrogen therapy was the obvious choice. The dosages prescribed initially were based on the schedule of estrogen therapy proposed by Smith[83] for the complications of pregnancy. The amounts had to be increased in the treatment of the severer types of angiectids. This can be correlated with our previously mentioned findings, namely, the association of the lowest estrogen titers in the series with the severest type of angiectid. Treatment of the 15 patients with orally administered female sex steroid hormones was associated in all cases with complete relief from pain and disability and a loss of the warmth, tenderness, and tension of the angiectids. Diethylstilbestrol § in dosages varying from 50 to 150 mg. daily proved effective in 13 of the 15 patients, including 1 patient in the group with a normal assay level. Relief was noted 24 to 72 hours after the onset of therapy. The patient with normal estrogen and subnormal pregnanediol levels did best with 100 mg. of oral progesterone || daily. In the remaining case (subnormal estrogen) the only effective therapy consisted of the oral use of both female sex hormones. Therapy in each instance was continued to the onset of labor, which was normal, as was delivery. There were no abnormal variations in labor, delivery, or the expected dates for such. After the termination of pregnancy, the angiectids disappeared completely in some, while others had faint intradermal vestiges.

The proposal by McCausland ¶ that an excess of circulating progesterone was the cause for varicose veins of the lower extremities complicating pregnancy has been widely accepted.# He reported a group of pregnant women with varices to have endocrine disorders on the basis of their variations from normal in body build, hair distribution, and menstrual history. McCausland claimed that the patients had an excess of circulating progesterone because they had a very low incidence of abortions and it was progesterone that had been reported to be the hormonal preventive of abortions.

# References 3, 5, and 13 to 25.

* References 2, 4, 6, 8, and 26 to 42; Eastman, N. J., in discussion on Hamilton.[3]

† References 4, 8, 26, 36, and 37.

‡ References 6, 28, 36, and 37.

§ Provided by E. R. Squibb & Sons, New York, as Stilbetin.

|| Provided by the Schering Corporation, Bloomfield, N. J., as ethisterone (Pranone).

¶ References 23, and 35.

# References 7, 8, 23, 36, and 37.

He also presumed that progesterone relaxed the walls of the veins, because the physiologic increase in progesterone during pregnancy had been reported to be a relaxant of the smooth muscle walls of the uterus, urethra, and intestine. McCausland concluded, therefore, that an excess of progesterone overly relaxed the walls of the veins to produce the varicosities. Not only have the clinical and laboratory findings of the present study differed with his theory, but progesterone has been found to have little, if any, effect upon the tonus of the blood-vessel walls.*

The clinical and laboratory findings in the present study associated the angiectids with variations in the amounts of circulating estrogen and progesterone. The symptomless angiectids were correlated with low normal amounts of the two hormones, as determined by assay methods. When the angiectids were painful, the levels of the hormones were subnormal. The degree of pain frequently was related to the level of the subnormal estrogen titer. The angiectids appeared shortly after the onset of pregnancy and disappeared with delivery, at which times the amounts of estrogen and progesterone change markedly. After delivery the intradermal vestiges of the angiectids only varied premenstrually, at which time they somewhat simulated the symptomatic angiectid of pregnancy. To these findings may be added our previously mentioned observation of the effectiveness of the female sex hormones in the treatment of the angiectids in a preliminary small group of patients.

The known increase in the amounts of estrogen during pregnancy and the reported vasodilating capacity of estrogen [44] may be reconciled with our findings which indicate that the hormonal factor was a disturbance in the estrogen-progesterone balance. Relative to the vasodilating action of estrogen, only the capillaries and venules are affected by the hormone.

The present study has added to the evidence of a relationship between the vascular system and the female sex hormones.†

Experimental studies have indicated that progesterone has a questionable vasodilating effect, whereas estrogen caused a marked, though temporary, vasodilation of the capillaries and venules.‡ The first clinical indication of the association § was noted in the initial report on Raynaud's disease. Raynaud found that one of the first signs of pregnancy in a patient with the disorder was a loss of the complaints due to the disease. Others || have observed the cyclic changes in the color and vascularity of the nasal mucosa during the menstrual cycle and during pregnancy. Palmar erythema and the acquired cutaneous arterial "spider" of the upper body have been assumed to be due to the physiologic increase in estrogens during pregnancy or to be due in patients with liver disorders to elevated estrogen levels caused by the disturbance of estrogen metabolism by the liver disease.¶

McGrath # and others * utilized the vasodilating capacity of the estrogens successfully in the treatment of peripheral vascular disorders. Its value also was reported in some patients with essential hypertension and angina pectoris.†

The laboratory studies that demonstrated the vasodilating effect of estrogen upon the blood vessels of the endometrium formed the basis for an accepted theory of menstruation.‡ The observations also led some to feel that the therapeutic effectiveness of estrogens in gynecologic disorders and complications of pregnancy in some instances was due to the vasodilating effect of the hormone.§ Our evidence for such will be published at a later date.

## SUMMARY

The superficial varicose veins of the lower extremities complicating pregnancy, which

* References 43 and 45.

† References 43 to 66.

‡ References 44, 45, and 48 to 66.

§ References 46 and 47.

|| References 52, 62, and 66.

¶ References 64, and 67 to 70.

# References 47, 71, and 72.

* References 46 and 74.

† Refrences 74 to 79.

‡ References 44, 45, 48, 49, 54, 55, 56, 62, 80, and 81.

§ References 58, 62, and 82; Smith, O. W., in discussion on Page.[84]

have been identified universally as classical superficial saphenous varicosities, were found to consist of two well-differentiated venous disorders. One is the mentioned saphenous varix. The other, which is limited to the pregnant state, had not been described previously and has been termed "angiectid" by us. The angiectid was present in 10% of a group of clinic patients and was nine times more frequent than the saphenous varicosity.

Many controveries have been due to the past failure to recognize the angiectid, which differs markedly from the saphenous varicosity. There is no resemblance between the well-known long, cord-like, subcutaneous saphenous varix and the angiectid, which is a small, intradermal, raised, sharply circumscribed mass of bluish vessels. The saphenous varix occasions fatigue and heaviness of the leg, which contrasts sharply with the pain and resultant disability that the angiectid causes in most cases.

In contrast to the saphenous varix, many clinical features of the angiectid indicated that an imbalance of estrogen and progesterone may be a factor in the formation of the angiectid. Hormone assays in 21 patients with angiectids demonstrated normal amounts of serum chorionic gonadotropin and urinary 17-ketosteroids and variations in the titers of serum estrogen and urinary pregnanediol. The six women with symptomless angiectids had consistently low normal amounts of estrogen and pregnanediol. Of the 15 patients with painful, disabling angiectids, 14 had subnormal amounts of estrogen and/or pregnanediol. Severe pain and marked changes in the angiectid were associated with the lowest subnormal estrogen levels. The results of the hormone assays in four patients with saphenous varices were normal.

Pain and disability due to the angiectids disappeared in each of the 15 patients with the use of orally administered female sex hormones. Diethylstilbestrol alone was effective in 13 cases.

The comment offers an explanation for the disputes related to the treatment and other aspects of the so-called varicose veins of the lower extremities complicating pregnancy, indicates fallacies in the claim that a high circulating level of progesterone is the cause of such varices, notes the likely role of the female sex hormones in the genesis of the angiectid, and further indicates a relationship between the female sex hormones and the vascular system.

Drs. Lewis C. Scheffey, Thaddeus L. Montgomery, and A. E. Rakoff cooperated in these studies, and Mr. Allan Hancock, F.B.P.A., produced the photograph.

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# Percutaneous Transhepatic Cholangiography

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Visualization of the hepatic and biliary ducts is difficult to obtain before operation when the gall bladder has either been removed or does not function. The use of sodium iodipamide (Biligrafin) by intravenous injection is sometimes satisfactory but is not satisfactory in the presence of jaundice and liver failure. A needle may, however, be introduced into the hepatic ducts by passing it through the skin and the liver, and when the duct system has been entered and bile aspirated, idopyracet (Diodone) may be injected and will give a satisfactory visualization of the ducts. This method has been used in a number of cases and with proper technique will show the site and nature of the obstruction; it also allows the pressure in the bile ducts to be recorded, and specimens of bile taken for chemical and bacteriologic investigation.

Carter and Saypol (1952), with the patient under general anesthesia, injected ducts in the left lobe of the liver and obtained a cholangiogram. Nurich, Patey, and Whiteside (1953) using local anesthesia introduced a needle at a point close to the right costal margin, and 3 to 5 cm. from the ensiform cartilage and obtained satisfactory cholangiograms.

Indications:

1. To investigate the cause of biliary symptoms after cholecystectomy
2. To diagnose the presence and site of a carcinoma affecting the biliary system
3. To demonstrate the presence, location, and number of calculi in the bile ducts
4. To measure the pressure of bile in the biliary system when spasm of the sphincter of Oddi is suspected and the investigation of the use of antispasmodic drugs

Submitted for publication July 8, 1955.

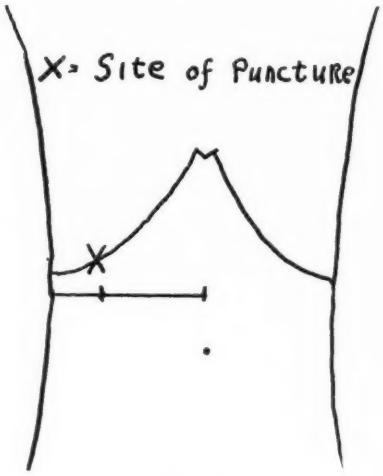


Figure 1

5. To diagnose the presence of obstructive jaundice as opposed to nonobstructive jaundice

TECHNIQUE

The method used in this series was to have the patient lying on the x-ray table and to make a subcutaneous procaine wheal in the skin just below the costal margin at the junction of the outer and middle third of a line joining the midline to the lateral border of the abdominal wall.

The subcutaneous fat, muscles and fascia are infiltrated and a needle 15 cm. long and 1 mm. in diameter is introduced at an angle

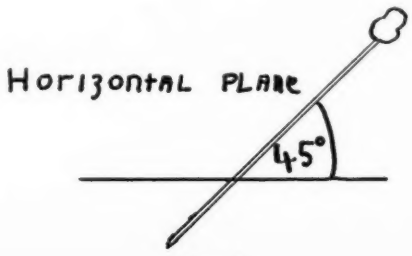


Figure 2

of 45 degrees to the horizontal plane and at an angle of 15 degrees to the inner side of the vertical plane.

The needle passes through the abdominal wall and enters the peritoneal cavity where the liver is usually in contact with the anterior abdominal wall.

The patient should now be instructed to take as shallow breaths as possible so as to reduce the excursion of the liver and on no account to cough. The needle is pushed on, in an upward and slightly internal direction for about 4 in. (10 cm.) depending upon the thickness of the abdominal wall. A record syringe allows frequent aspirations to be

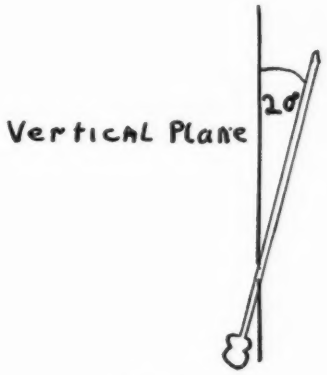


Figure 3

made and it will often happen that blood is aspirated from the venous sinuses. The needle should, however, be pushed on and its whole length introduced. Bile is usually aspirated about after 12 cm. have been inserted. In cases of jaundice with obstruction the puncture is usually easily made and the bile can then be collected for culture and biochemical investigation. The pressure may also be measured by attaching a simple graduated glass tube as used for measuring cerebrospinal fluid pressures. When the pressure is high it is advisable to aspirate 30 cc. or more of bile in order to obtain complete filling of the duct system. Eighteen cubic centimetres of 50% iodopyracet is then injected and anteroposterior and lateral x-ray photographs of the liver are taken.

DIFFICULTIES AND DANGERS

Some pain is often experienced by the patient when the needle enters the capsule of the liver, and he should be warned of this beforehand to prevent him taking a sudden deep breath; though with the use of a fine, flexible needle no trouble has been experienced from sudden inspiration or coughing. It is most important to use a narrow-gauge needle that will bend easily.

The aspirated blood in the syringe may make the detection of bile difficult and the syringe should be detached and washed out with saline. No trouble has been encountered from intrahepatic or abdominal hemorrhage and indeed when operation has been performed immediately after the cholangiography, in some cases, it has been difficult to see the puncture mark on the liver.

If bile cannot be aspirated during the introduction or withdrawal of the needle, the needle should be reinserted in a slightly different place.

The chief danger is to puncture the gall bladder, as bile will leak through the puncture wound and give rise to bile peritonitis. Death has been recorded from this mishap by another investigator. In one case, in my series, when the gall bladder was known to have been punctured, instructions were given that should abdominal pain occur, immediate cholecystectomy should be performed. Pain did in fact occur, and the gall bladder was exposed eight hours after the puncture. There was a biliary leak and local bile peritonitis, but the patient made an uninterrupted recovery after cholecystectomy. Now, when preoperative percutaneous cholangiography is to be performed in a patient whose gall bladder is present, the operation is arranged to be done on the same day.

If bile is aspirated after the needle has only been inserted for a short distance and the gall bladder is thought to have been punctured, as much bile as possible should be aspirated. About 5 cc. of iodoypracet may then be injected to confirm the position of the needle and the needle may then be pushed on through the hepatic surface of the gall

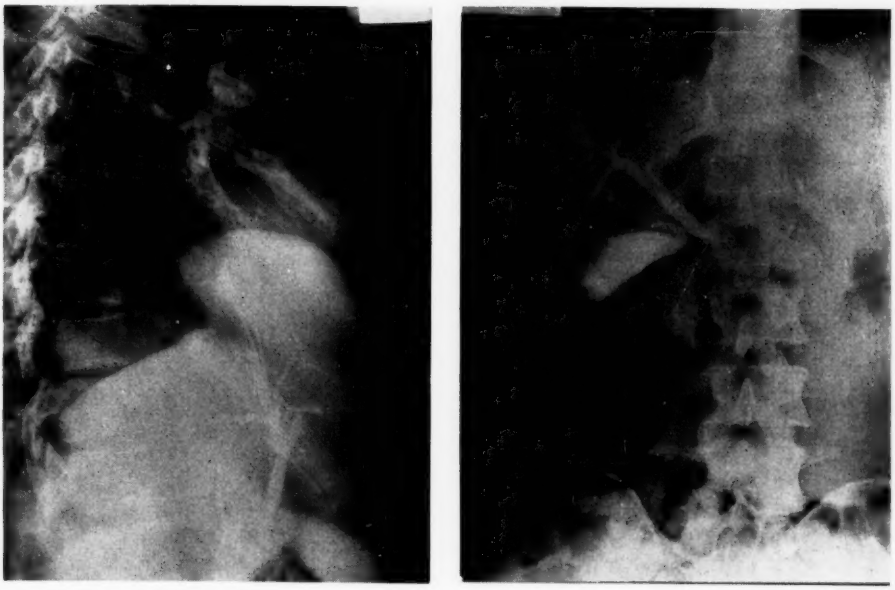

Figure 4

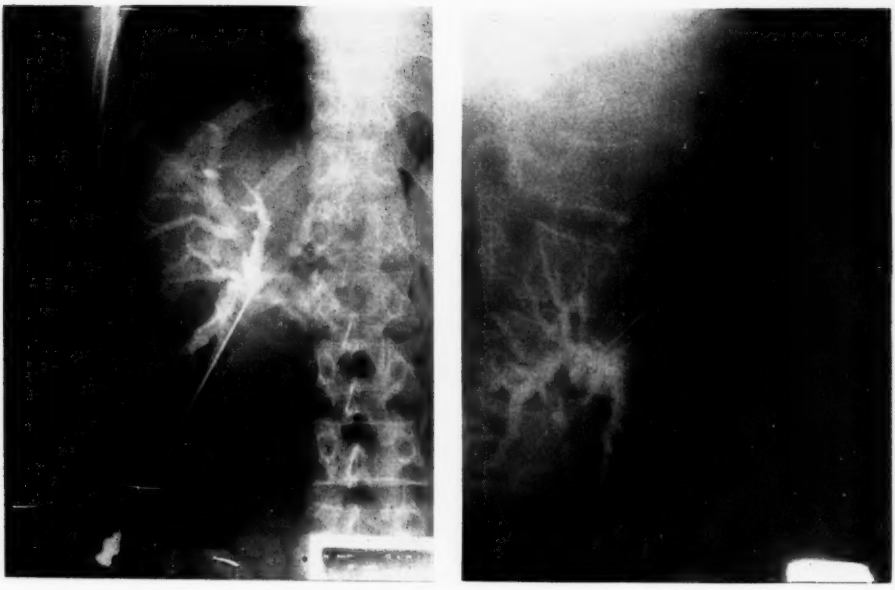

Figure 5

Figure 6

Figure 7

Figure 8



Figure 9

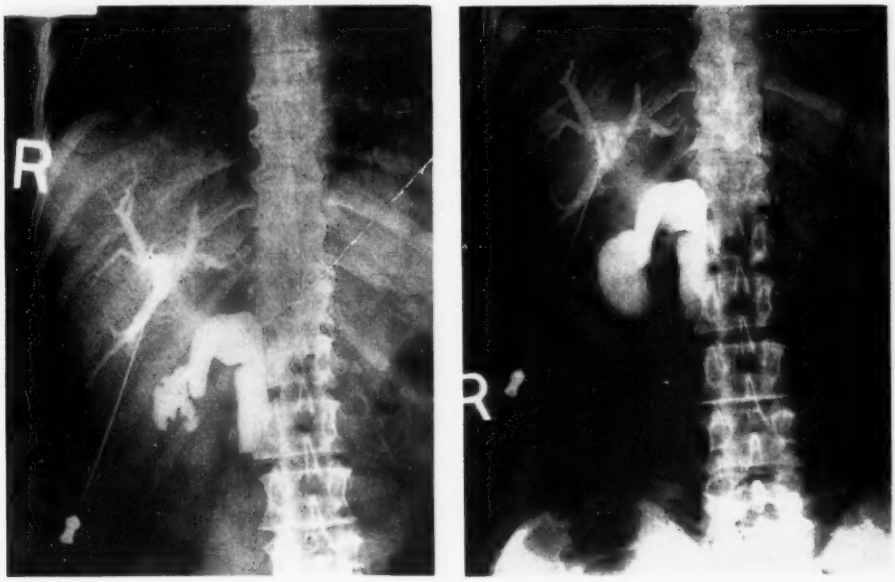


Figure 10

bladder where a main duct will probably be entered. In this case, operation should, of course, be performed during the next few hours.

When there is complete or almost complete biliary obstruction some 30 cc. of bile should be aspirated as the iodopyracet does not mix with the bile and the whole system will not be properly visualized. This occurred in Case 2 in which the common bile duct was obstructed 2.5 cm. from the ampulla of Vater by a carcinoma in the head of the pancreas. Although good filling of the biliary tree was obtained, failure to visualize the common bile duct led to an error in diagnosis; the obstruction being thought to be due to secondaries in the porta hepatis.

Puncture of colon or small bowel, if it ever occurred, has never given rise to any untoward symptoms, though it could do so if intestinal obstruction were present.

## REPORT OF CASES

Case 1.—F. R., a man, aged 47. Resection of pancreas for carcinoma Nov. 14, 1951. One year later complained of pain in the back and vomiting. No jaundice. Dec. 23, 1952: percutaneous cholangiography. A specimen of aspirated bile gave a scanty growth of Escherichia coli. Twenty cubic centimeters of 50% iodopyracet was injected and the cholangiograms showed good filling of hepatic ducts and gall bladder with dilated common bile duct but free passage of dye into jejunum and patency of the end-to-end choledochojejunal anastomosis. The patient died March 6, 1954, from multiple recurrences.

Case 2.—E. H., woman, aged 56. History of jaundice and loss of weight for one month. Percutaneous cholangiography showed good filling of the intrahepatic ducts, but the common hepatic was only partly filled and neither the gall bladder nor common bile duct was visualized. Incomplete filling led to a wrong diagnosis of extrabiliary pressure from secondaries in the portal fissure. This was due to an error of technique because not enough bile was aspirated and the iodopyracet does not mix with the bile. At operation a carcinoma of the pancreas was found and the common bile duct although grossly dilated was only obstructed at the ampulla of Vater.

Case 3.—K. R., man, aged 45. History of jaundice and loss of weight for two months. Attempted percutaneous cholangiography resulted in puncture of the gall bladder and films showed dye in gall bladder and a subcapsular infiltration. At operation a few hours later there was leakage of bile from the gall bladder and inoperable carcinoma of the

pancreas was present. Cholecystectomy was performed and the common bile duct drained.

CASE 4.—A. H., man, aged 65. Cholecystectomy 1951. Common bile duct explored. No calculi found; drainage of duct. Well for two years, then obstructive jaundice. Percutaneous cholangiography gave good visualization of the biliary tree, except the left lobe of the liver, and showed a stone impacted at the ampulla of Vater. Only one stone was present and at operation four hours later the stone was removed from the ampulla. Confirmatory postoperative cholangiography showed free passage of bile.

CASE 5.—E. D., woman, aged 76. General condition very poor from progressive jaundice and loss of weight. Percutaneous cholangiography, 12 cc. 50% iodopyracet was injected. Films were unsatisfactory as the patient moved. Fifty cubic centimeters of bile were aspirated, and 18 cc. of iodopyracet injected. Films showed filling of the intrahepatic ducts, but a block in the common bile duct. A diagnosis of carcinoma of the common hepatic duct was made. The findings were confirmed at laparotomy when the duct was drained. The gall bladder was found to be packed with stones and functionless. The patient never recovered consciousness, and autopsy and microscopic examination confirmed the diagnosis.

CASE 6.—D. N., woman, aged 41. Six weeks' history of pain in the right hypochondrium and occasional vomiting. Short periods of slight jaundice one month previously. Two attempts at bile duct puncture failed. At third essay bile was aspirated at a depth of 7 cm., suggesting that the needle was in the gall bladder. Forty cubic centimeters of bile was aspirated and 8 cc. of iodopyracet injected. The needle was then pushed on into the liver for a further 5 cm. and a bile duct was entered. Eighteen cubic centimeters of iodopyracet was injected. Films showed the whole of the biliary tract outlined and the presence of one calculus in the common bile duct and numerous calculi in the gall bladder. The dye had also entered the duodenum. At operation one hour later there was no bile leakage and cholecystectomy was performed and a stone removed from the common bile duct. A small puncture point was visible on the liver and the needle had in fact transfixed the gall bladder on its hepatic surface, so no bile could have leaked into the peritoneal cavity.

## CONCLUSIONS

Percutaneous cholangiography is of value in the preoperative investigation and diagnosis of diseases affecting the biliary tract. It should, however, be carried out in a surgical unit when immediate operation can be performed should the gall bladder be inadvertently punctured.

Dr. Lavelle, Director of the X-ray Department, and his staff at St. Helier Hospital, Carshalton, Surrey, gave technical help and cooperation.

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# Mortality Rate of Cholecystectomy with a Curarizing Agent

**Study in a Series of Cases**

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**and**
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In a recent report Beecher and Todd [1] stressed the fact that the mortality rate was higher in patients who receive curare than in those who did not receive curare. They further stated that "the best answers to certain kinds of questions can be obtained by limiting the inquiry in future studies to certain kinds of severe test situations as anesthesia for common bile duct surgery."

Orr [2] in a study of the mortality rate in a series of 558 cholecystectomies reported a mortality rate of 2.8%. He further stated that "skilled anesthetists have contributed their part in reducing the death rate."

This review of 563 cholecystectomies is a continuation of Orr's original report and covers a period from Jan. 1, 1946, to Dec. 31, 1954. In this series of cases a curarizing agent was used in 440 cases.

The diagnosis of gall-bladder disease is usually not difficult; but failure to recognize or estimate the importance of associated disease such as coronary sclerosis, myocardial insufficiency, chronic respiratory disease, and associated pathology may be difficult, and these are important in an evaluation of the surgical mortality.

Accepted for publication July 27, 1955.

From the Department of Anesthesiology, University of Kansas Medical Center (Dr. Lorhan).

Read before the Section on Anesthesiology at the 104th Annual Meeting of the American Medical Association, Atlantic City, June 10, 1955.

TABLE 1.—*Operation and Relation to Mortality Rate*

| Type of Operation | No. of Operations | No. of Deaths | Per Cent Mortality |
|---|---|---|---|
| Cholecystectomy | 294 | 4 | 1.36 |
| Cholecystectomy and appendectomy | 81 | 0 | .... |
| Cholecystectomy and choledochostomy | 114 | 5 | 4.38 |
| Cholecystectomy, choledochostomy, and appendectomy | 21 | 0 | .... |
| Cholecystectomy and other major and minor operations | 53 | 1 | 1.88 |
| | 563 | 10 | 1.77 |

However, this discussion concerns itself as to whether or not a curarizing agent is a contributing factor in the cause of death following surgery upon the gall bladder.

In Table 1 are listed the operations and number of deaths following each procedure. When choledochostomy is added to the procedure it will be seen that the mortality is increased. This increase in mortality has been substantiated by others.

The ratio of men to women was approximately one to four. The average age of men was 55 and of women 49.4 years. These ages are comparable to those of Orr's patients. The average age for men at death was 70.15 years and for women 62.5. These figures differ from Orr's, as will be seen in Table 2. It has been admitted by surgeons [3]

TABLE 2.—*Comparison of the Mortality Rates in Cholecystectomies with Those of Orr (in Parentheses)*

| Sex | No. of Patients | Average Age | Average Age at Death | No. of Deaths | Per Cent Mortality |
|---|---|---|---|---|---|
| Males | 150 (93) | 55 (52.6) | 70.15 (55) | 4 (7) | 2.66 (7.5) |
| Females | 413 (465) | 49.4 (48.1) | 62.5 (52) | 6 (9) | 1.45 (1.8) |
| Total | 563 (558) | | | 10 (16) | 1.77 (2.2) |

TABLE 3.—*Anesthetic Agents Used with Curarizing Agent*

| | No. of Cases | No. of Deaths | Per Cent Mortality |
|---|---|---|---|
| Thiopental-nitrous oxide | 150 | 2 | 1.33 |
| Thiopental-nitrous oxide-ether | 32 | 1 | 3.12 |
| Cyclopropane | 127 | 4 | 3.15 |
| Cyclopropane-ether | 85 | 2 | 2.35 |
| Tribromoethanol (Avertin)-nitrous oxide-ether | 1 | .. | .... |
| Thiopental-ethylene | 1 | .. | .... |
| Multiple agents | 40 | .. | .... |
| Spinal—tetracaine (Pontocaine), thiopental | 1 | .. | .... |
| Spinal—tetracaine, cyclopropane | 2 | .. | .... |
| Continuous spinal—procaine thiopental | 1 | .. | .... |
| Total | 440 | 9 | 2.05 |

that the operative risk of the aged patients is increased when they are undergoing gallbladder surgery. Since so many patients with disease of the biliary tract fall in the aged group when respiratory, cardiovascular, renal, and liver pathology are prevalent, it is imperative then that all patients have a careful physical examination before surgery. Furthermore, if Beecher's hypothesis is correct, that curare is the responsible factor in the mortality rate, we should have had a higher percentage mortality than in Orr's series. In 563 cases, the over-all mortality rate was 1.77%, as compared with 2.8% in 558 cases. Again, if we were to include the 123 cases of our series who did not receive a curarizing agent the mortality would still be 2.56%, whereas the mortality in 440 cases receiving a curarizing agent is only 2.04%.

The anesthetic agents used varied with the individual administrator. Thiopental sodium (Pentothal Sodium)-nitrous oxide-oxygen was the principal agent used in 150 cases, and cyclopropane, in 127 cases. An ineffective spinal anesthetic agent was supplemented with another agent in four cases. It will be seen from Table 3 that, the operation with cyclopropane anesthesia when complemented with a curarizing agent had a percentage mortality of 3.15, whereas with thiopental sodium-nitrous oxide-oxygen anesthesia the mortality was only 1.33%.

In this series of 440 cholecystectomies with a curarizing agent, there were 10 deaths. There were 10 autopsies. The cause of death, with the duration of operation, anesthetic agent, and physical status, is listed

TABLE 4.—*Deaths Following Surgery*

| | Operation | Sex | Age, Yr. | Physical Status | Anesthetic | Curarizing Agents, Mg. | Duration of Anesthesia, Hr. | Cause of Death |
|---|---|---|---|---|---|---|---|---|
| 1 | Cholecystectomy | M | 74 | 3 | Cyclopropane | Curare 6 | 3 | Cerebral vascular, 24th postoperative day |
| 2 | Cholecystectomy | F | 71 | 3 | Thiopental-nitrous oxide | Curare 17 | 2 | Auricular fibrillation, bronchopneumonia, 5th postoperative day |
| 3 | Cholecystectomy | F | 63 | 4 | Cyclopropane-ether | Curare 10.5 | 2 | Cardiac failure, 2d postoperative day |
| 4 | Cholecystectomy | M | 62 | 4 | Cyclopropane | Curare 18 | 1½ | Peritonitis, 3d postoperative day |
| 5 | Cholecystectomy and choledochostomy | F | 68 | 2 | Thiopental-nitrous oxide-ether | Curare 6 | 2 | Electrolyte imbalance, 7th postoperative day |
| 6 | Cholecystectomy and choledochostomy | M | 79 | 2 | Thiopental-nitrous oxide | Succinylcholine 500 | 3 | Hemorrhage, peritonitis duodenal fistula, 6th postoperative day |
| 7 | Cholecystectomy and choledochostomy | F | 65 | 3 | Cyclopropane | Succinylcholine 650 | 1 | Subphrenic abscess, fecal fistula, 14th postoperative day |
| 8 | Cholecystectomy and choledochostomy | M | 71 | 3 | Cyclopropane | Curare 6 Decamethonium bromide (Syncurine) 2 | 1 | Hepatitis, 30th postoperative day |
| 9 | Cholecystectomy and repair of ventral hernia | F | 43 | 4 | Cyclopropane | Curare 10.5 | 2 | Atelectasis, 4th postoperative day |

TABLE 5.—*Mortality Rate*

| | | Per Cent Mortality |
|---|---|---|
| Total cases with curarizing agents... | 440 | |
| Deaths attributed to anesthesia..... | 1 | 0.227 |
| Deaths not related to anesthesia..... | 8 | 1.8 |

in Table 4. In only one case may death be directly attributed to the anesthetic. This patient died of bilateral atelectasis on the fourth postoperative day. The physical status was 4. She was markedly obese, weighing 259 lb., and had had hypertensive cardiovascular disease. There was such an imposing array of pathologic findings in most of the cases that it was difficult to decide which disease was the primary cause of death. Bronchopneumonia was present in two cases but probably was the primary cause of death in one case.

In Table 5 is the per cent mortality rate of the 440 cases which received a curarizing agent. The mortality rate directly attributable to the anesthetic is 0.227%, whereas the number of deaths not related to anesthesia is 1.8%. If we were to consider all the deaths as being directly attributable to the use of a curarizing agent, the mortality rate would only be 2.045%. This figure agrees with that of Glenn and Hays,[4] who found the mortality rate to be 2.5%. In the geriatric patient, 65 years of age or over, the over-all mortality rate is approximately 6.5%. Ochsner [5] in a recent report gives the mortality rate in a series of cholecystectomies as 7.5%.

In Table 6 are listed the various curarizing agents used. *d*-Tubocurarine chloride was used in 272 cases, with six deaths, or a 2.20% mortality rate; succinylcholine (Anectine) in 94 cases with two deaths, or a mortality rate of 2.12%.

TABLE 6.—*Curarizing Agents*

| | No. of Cases | No. of Deaths | Per Cent Mortality |
|---|---|---|---|
| Curare | 272 | 6 | 2.20 |
| Gallamine triethiodide (Flaxedil) | 6 | 0 | .... |
| Decamethonium bromide (Syncurine) | 44 | 0 | .... |
| Succinylcholine chloride | 94 | 2 | 2.12 |
| Combined | 24 | 1 | 4.66 |
| Total | 440 | 9 | 2.05 |

An attempt was made to evaluate the factor of the qualifications of the operating personnel. In Table 7 is tabulated the mortality rate in relation to the participation of a staff surgeon, resident, staff anesthesiologist, and resident in the operations. The highest mortality occurred when the resident surgeon did the operations. The staff anesthesiologist administered 142 anesthetics, with two deaths, for a mortality of 1.40%. This is explainable by the fact that a staff anesthesiologist always gave the anesthetic whenever the gravity of the case warranted good medical management. It is of interest to note that when the resident surgeon performed the operation the mortality rate was 2.35%, whereas with a staff surgeon the mortality was only 1.13%. This would raise the question as to what part the experiences of the surgeon had in affecting the over-all mortality rate in patients undergoing gall bladder surgery.

TABLE 7.—*Relation of Surgeon and Anesthetist to Mortality Rate*

| | Surgeon | | Anesthesiologist | |
|---|---|---|---|---|
| | Staff | Resident | Staff | Resident |
| Number of cases.. | 350 | 213 | 142 | 384 |
| Number of deaths. | 4 | 5 | 2 | 7 |
| Per cent mortality | 1.13 | 2.35 | 1.40 | 1.82 |

The validity of Beecher's statement that the mortality rate is higher in patients who receive curare is questioned. It is realized that muscle relaxants are not innocuous and may occasionally be the responsible factor in the cause of death of a patient. However, before condemning an agent and relegating it to the side lines, one must ascertain whether or not that agent per se has deleterious effects upon the patient. All too frequently anesthetic agents have been blamed for causing the death of a patient, whereas the surgeons and the anesthesiologist should be considered as the primary cause of death.

SUMMARY

The mortality rate in a series of 440 cholecystectomies receiving a muscle relaxant is reviewed.

The mortality rate directly attributable to the anesthetic was 0.227%; the rate in 440 cases receiving a curarizing agent was 2.045%.

The mortality rate in 563 cases was 1.77%.

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# Strangulated Right-Sided Diaphragmatic Hernia

**Report of a Case**

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Strangulation of a diaphragmatic hernia is, in general, a rather rare phenomenon, in contrast to the common occurrence of strangulation in other types of hernia. An explanation for this is that the great majority * of diaphragmatic hernias are of the esophageal hiatus type, in which usually the stomach is the only viscus herniated. The heavy muscular wall and excellent blood supply of the stomach protect it from strangulation, though incarceration is common enough. In the congenital group of diaphragmatic hernias, including the pleuroperitoneal type (Bochdalek's), congenital absence of the posterior diaphragm, and the subcostosternal type (foramen of Morgagni), though various viscera may be extruded into the chest, strangulation is still uncommon. Intestinal obstruction, however, is a real possibility, especially in the large defects of the newborn.†

Far exceeding the expected incidence of strangulation, according to its proportion to other diaphragmatic hernia types, is the traumatic type. In a collection of 43 cases of strangulated diaphragmatic hernia, Carter and Giuseffi [6] found that only 4 occurred in congenital types; the remainder were traumatic. This may be due to the adhesions and scarring incident to the trauma involved: frequently, penetrating wounds, compression injuries, or necrosis from tube drainage of empyema or subdiaphragmatic abscesses. As would be expected, the left side of the diaphragm is the side most frequently involved in traumatic hernia, the liver protecting the right side from herniation in most cases.‡ The case presented here represents an example of strangulating right-sided diaphragmatic hernia of the posttraumatic type and is of especial interest in that the trauma preceded the acute symptoms by 25 years and because of the unusual content of the incarcerated mass.

## REPORT OF A CASE

A 74-year-old white man was admitted to the hospital on April 28, 1955, because of severe generalized abdominal pain. On April 27, after his evening meal, he had some abdominal cramping, relieved shortly by defecation. On the day of admission he had felt well until the evening, when, after moving some furniture, he was rather suddenly seized by severe pain throughout his abdomen, which varied in intensity every few minutes. There was some nausea but no emesis. He had had three loose stools that day.

His past history included a more or less chronic asthma, and he had been studied 2 years before for heart disease because of occasional precordial pressure, with essentially negative results. There was no history of chronic indigestion, though subsequent to his operation he admitted that his precordial pressure was occasioned by reclining after a large meal. History of an automobile accident 25 years before in which his left chest was injured was also obtained on questioning postoperatively.

His general appearance was that of a man of about 70 years, of average weight and build, in great distress. He was sitting up and said the pain was worse when lying down. He was unable to localize his pain. The abdomen was slightly dis-

Submitted for publication June 30, 1955.

* References 1 and 2.

† References 3, 4, and 5.

‡ Clagett, O. T., personal communication to the author.

tended, and a moderate rigidity all over seemed voluntary. There was tenderness in all parts of the abdomen, but it was worse in the lower half. There was no rebound tenderness. Peristalsis was active, with occasional rumbling, loud borborygmi. Tympany was noted to extend over the liver area. There were coarse rales and some dullness at the base of the right lung posteriorly. The heart sounds were normal, though distant. The rest of the examination, including a rectal investigation, was negative. It was noted that femoral pulses were good and that there were no herniae or operation scars apparent.

On his admission the laboratory reported 12.45 gm. of hemoglobin and a white-blood-cell count of 9150, of which 55% were segmented neutrophiles, 10% stab forms, and 35% lymphocytes. Urinalysis was negative. An x-ray of the abdomen revealed loops of distended small bowel in the right upper quadrant, extending as high as the diaphragm. The top of the diaphragm did not appear on the film.

The patient was observed for about 4 hours with no improvement and then was operated upon for small-bowel obstruction, possibly closed-loop type.

*Operation.*—A spinal anesthetic was given, but as the incision (right rectus) was extended upward it proved inadequate and an inhalation anesthetic was given. Bloody fluid was found in the abdomen along with distended small bowel obstructed by a strangulating hernia in the posterior portion of the right diaphragm. This was found to be approximately 10 by 6 cm. in size, and by slipping a tube into the defect most of the incarcerated viscera could be withdrawn. A large amount of omentum was fixed in the hernia and had to be resected. Most of the right colon was in the chest, as were as a few feet of distended ileum. All was viable. A large Meckel diverticulum was in the herniated portion. The right lobe of the liver, which was atrophic, protruded through the diaphragm to the extent that the gall bladder, shrunken and full of stones, was just within the chest. The liver was apparently fixed in the chest, forming the medial and posterior walls of the hernia opening. For this reason a satisfactory repair was impossible from below. As the patient aspirated vomitus during the operation and was a poor risk, the operation was concluded and vomitus aspirated from the trachea by bronchoscope. There was no collapse of the lung as adhesions had apparently formed a false sac.

Postoperatively the expected ileus produced a very minor wound disruption in spite of constant suction and use of an oxygen tent. However, the patient had no respiratory complications and made a good recovery.

## SUMMARY

A case is presented having the unusual condition of a right-sided diaphragmatic traumatic hernia. The extrusion of the right lobe of the liver into the chest prevented that organ from performing any obturator function, thus allowing the other viscera to ascend into the chest and strangulate. The presence within the chest of a Meckel diverticulum and a gall bladder full of stones was a bizarre coincidence.

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# Cervical Thymus

**Report of a Case**

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Persistence of the thymus gland in the neck is extremely rare. The usual case reported is that of a hypertrophied thymus in its normal position in the mediastinum, with a portion projecting upward into the root of the neck.* Two instances of thymic cyst have been found in the neck.† Microscopic rests of thymic tissue in or near the parathyroid glands have been identified and are explained as the result of development from contiguous embryological anlage.‡ In the following case, a tumor of the neck was removed and was found to be morphologically identical with the thymus. We have not been able to find a similar case recorded.

## REPORT OF CASE

A 3½-year-old white boy was admitted to the Barnert Memorial Hospital on June 23, 1954. His mother had first noticed a small lump in the left side of his neck about 18 months previously. The lump had gradually increased in size. Four months before admission, during a febrile illness, the mass had become larger, tender, and tense, and then had receded to its previous state after a series of penicillin injections. Hoarseness had been noted for three weeks. There was no dyspnea or dysphagia. The past history, including birth and early development, was normal.

Physical examination showed an alert, well-developed, and well-nourished boy with no abnormalities except a soft cystic swelling in the left side of his neck, below the level of the thyroid cartilage. The mass appeared to be superficial but could not be accurately defined. It was estimated to be 1 to 1.5 in. in diameter. X-ray of the neck showed a soft tumor mass displacing the trachea to the right and posteriorly. There was no involvement or displacement of the esophagus. The blood count was normal. The tentative diagnosis was branchiogenic cyst.

At operation a low transverse incision was made. The platysma and cervical fascia were incised and the left strap muscles clamped and divided. Between these muscles and the carotid sheath there was a large encapsulated mass. It felt cystic and was

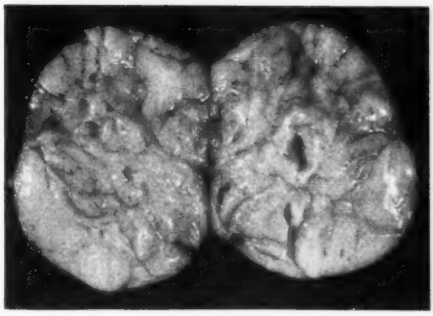

Fig. 1.—Gross appearance of the mass on section.

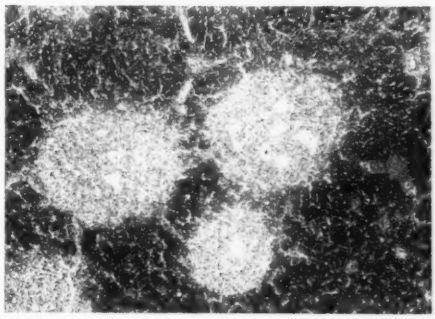

Fig. 2.—Microscopic appearance, showing indistinct separation of lobules, well-developed medullary zones, and numerous Hassall's bodies. Reduced about ¼ from mag. × 12.

Submitted for publication June 1, 1955.

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* References 1 and 2.

† References 3 and 4.

‡ References 5 and 6.

attached to the left paraesophageal tissue by a narrow vascular pedicle. The mass was freed by blunt dissection, and the pedicle was clamped and tied. Recovery was uneventful. On follow-up examination eight months after operation, the scar was soft and inconspicuous. There was no sign of recurrence of the mass. At this time, x-ray of the chest did not reveal a thymus shadow. There were no endocrine abnormalities; no muscle changes were seen.

The specimen weighed 85 gm., three and a half times the weight of a normal thymus in a 4-year-old child.[7] It was an encapsulated mass, moderately soft in consistency, measuring 8×6×4 cm. On cross section the parenchyma was lobulated, glistening, and grayish-tan in color, with pinhead-sized hemorrhagic areas.

Histologically, the individual lobules were large. The dividing septa were composed of a small amount of fibrous and fat tissue with many lymphocytes. The cortical areas were very wide, occupying more than half of the lobules; the medullae consisted of lymphocytes, reticulum cells, and mature Hassall's bodies.

In order to determine whether this mass represented hyperplasia or a tumor, the microscopic picture was compared with that of normal glands in children of similar age. Only cases of sudden or accidental death were reviewed to eliminate effect of "accidental involution."[8] Sections of this mass showed much wider cortical areas than normal glands and could not be distinguished microscopically from cases of marked hyperplasia, as observed in our sections from anencephalic monsters and from other known cases of thymic hyperplasia.§

## COMMENT

For the surgeon, the preoperative diagnosis of discrete lateral tumors of the neck can only be tentative and must be confirmed by exploration and pathologic examination. The long list of diagnostic possibilities has been tabulated by Lahey.[11] Cervical thymus must be appended to this list for consideration before operation.

For the pathologist, this case poses several interesting problems. The removed thymic tissue may be classified as one of the following types: (*a*) an accessory unilateral cervical thymus, (*b*) a unilateral thymus gland, undescended, or (*c*) a hyperplastic gland or a tumor. X-ray of the chest may be helpful. If thymic shadow is seen, it is proof of the presence of thymus in the mediastinum. Negative films, as in this case, do not exclude the existence of the gland in the chest. Considering the size and shape of the specimen as well as the history, we can assume that we are dealing with a benign thymic tumor having the histological structure of a hyperplastic gland.

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## SUMMARY

A case of cervical thymus removed surgically is presented.

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# Traumatic (False) Aneurysms

## The Hazard of Undue Delay in Surgical Treatment

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Many types of surgical procedures have been devised in an attempt to cure arterial aneurysms. Antyllus and Purmann were outstanding pioneers in this field. The technique devised by Hunter (proximal ligation of the artery) was practiced because of the dangers inherent in the direct surgical approach to an aneurysm. Matas performed the first endoaneurysmorrhaphy in 1888, and his work during the next decade, with that of Halsted, led to the modern development of the surgery of aneurysms.

### PATHOGENESIS OF TRAUMATIC ANEURYSMS

In the formation of a traumatic aneurysm there must be one constant condition. The injury to the vessel must be partial. Should complete severance of a vessel occur, retraction and clotting will take place. Often the presence of an aneurysm is not discovered for days or weeks. Many small leaks may seal off spontaneously and never break down. If there is partial division of an artery and there is persistent bleeding, a pulsating hematoma usually develops. The spasm in the vessel wall causes the rent to gape, and bleeding into the adjacent tissues continues. Bleeding ceases only when the pressure in the hematoma is equal to the arterial pressure within the involved vessel. The amount of bleeding and the size of the hematoma depend upon the resistance of the adjacent tissues and upon the size of the artery. The more tense the tissue planes, the smaller the hematoma. A clot may temporarily seal the gap in the vessel wall, only to give way days or weeks later. It is possible, even without an actual tear, for a contused portion of the vessel wall to give way and cause delayed hemorrhage. These aneurysms tend to bleed unexpectedly and profusely.

The central portion of the hematoma usually becomes liquefied, and a pulsation appears. Organization of the clot begins at the periphery so that within several weeks there is an endothelial-lined sac. Infection may develop and interfere with the deposition of firm fibrous tissue; hence, softening and rupture of the sac wall may occur. This hemorrhage may cause damaging pressure upon adjacent nerves and collateral blood supply. The pressure may be so extreme that ischemia and gangrene may develop. If the clot is absorbed and a sac membrane is formed, the lesion becomes a traumatic, or false, aneurysm. This differs from a true aneurysm in that the wall of the sac is not made up of the three layers of the normal vessel wall but only of lining endothelium and organized clot. Thus, the pulsating hematoma is the precursor of all traumatic aneurysms. Unlike acquired arteriovenous fistulas, in the case of traumatic aneurysms there is no communication with the adjacent vein.

### DIAGNOSIS

A traumatic aneurysm usually produces few symptoms. There may be moderate pain, symptoms of arterial insufficiency, or evidence of pressure on nerves. Severe, unremitting pain usually indicates an impending complication.

Physical examination usually reveals a tense, pulsating mass. A thrill may or may not be palpable, but a systolic bruit is in-

Submitted for publication June 3, 1955.
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variably present. There is usually no change in the temperature of the extremity. The distal pulses and blood pressure are usually unchanged, unless there is a large hematoma which mechanically occludes the artery. Digital compression of the proximal artery produces no slowing of the pulse rate or rise in blood pressure. The distal venous pressure usually shows no change from the normal.

The skin overlying a traumatic aneurysm may be shiny, firm, tender, and warm, thus presenting the clinical picture of an abscess. Preliminary aspiration of any suspicious mass should always be carried out. If blood is aspirated, provisional control of the major artery proximal and distal to the aneurysm must be obtained before direct entry is made into the lesion.

### INDICATIONS FOR OPERATION

The indications for operation upon traumatic aneurysms fall into the following general groups: (1) emergency indications, such as violent hemorrhage, (2) increasing tension within the sac, (3) marked, spreading infection, (4) evidence of progressive ischemia distal to the aneurysm, and (5) signs of progressive nerve damage.

There is general agreement that corrective operations for acquired arteriovenous fistulas should be postponed for three to six months from the time of injury, to allow an adequate collateral circulation to develop. There is a difference of opinion, however, as to when traumatic aneurysms should be operated upon. Reparative and restorative operations may be employed more frequently, and the dangers of pressure from a progressing hematoma will be minimized if immediate operation is carried out. On the other hand, immediate operation may be hazardous if there is a large hematoma or if the patient has just recovered from shock or other injuries. There is also a danger of infection, since an immediate operation is frequently conducted in a potentially infected field. Makins believes that operation should be postponed if the hematoma is of three days' duration or longer, unless significant hemorrhage or infection continues.

Although hazardous complications may occur at any time, an effort should be made to carry a limb long enough so that an effective collateral circulation can develop. In the presence of a traumatic aneurysm the collateral circulation is not greatly stimulated, since the artery usually is not occluded. The full arterial force is directed into the aneurysm, increasing the likelihood of rupture. A delay of from three to four weeks would seem desirable, however, to allow for the subsidence of all inflammation and the development of collateral circulation. During this period, careful observation is essential in order to detect any sudden complication. If pain is severe and unremitting, the danger of delay is considerable.

It may be very difficult to determine the adequacy of the collateral circulation prior to operation. If the vessel proximal to the site of injury is available for selective compression, the Moschcowitz-Matas test can be used.

Elevate the extremity to an angle of 30 degrees, compress the artery with the fingers just proximal to the point of injury and apply an Esmarch bandage from the fingers or toes up to the point of injury. After maintaining bandage and pressure for five minutes, release the bandage while maintaining digital pressure on the artery. The limb should flush down to the fingers and toes within, at most, three minutes, preferably within one minute.

This procedure is not feasible in many instances, but there are other guides as to the adequacy of collateral circulation. A diminished or absent arterial pulse in a warm or pink extremity is a good sign, since it is evidence that the collateral circulation is already effective. Whiteness of the limb is indicative of poor circulation. Nail-bed reactions to pressure and the rapidity of venous filling are valuable diagnostic tests. If these tests reveal an insufficiency of the collateral network, further delay of operation is indicated, except under urgent conditions.

### ROLE OF PRELIMINARY SYMPATHECTOMY

Interruption of the sympathetic pathways has been found useful in the treatment of an ischemic limb. Experience has supported the view that when ligation of a main artery in

an extremity is inevitable the risk of gangrene can be reduced by preliminary sympathectomy. Gage reported real improvement in collateral circulation in 15 patients with arterial and arteriovenous aneurysms after the injection or ablation of the regional sympathetic ganglia. Veal suggests that sympathectomy may prove of additional value in the future nutrition of a limb, because with a leg at rest in a warm environment the circulation may be adequate and the patient symptom-free, but it may be wholly inadequate under the stress of normal function or if there are fluctuations in environmental temperature. Schumacker suggests that sympathectomy may be of value in two ways: It may eliminate vasoconstrictor impulses and contribute to the maintenance of near-maximal circulation, and it may also aid in the development of new collateral channels through a reduction in peripheral resistance.

## OPERATIVE TECHNIQUE

At the time of operation for a traumatic aneurysm it is often impossible to repair the hole in the vessel, since it involves too much of the arterial circumference. Ligation in continuity should not be carried out. The use of tourniquets is not desirable, since the trauma involved may add to the vascular spasm which is present. A tourniquet may be set in place, to be tightened only if uncontrollable bleeding occurs. The incision should take into consideration all known collateral vessels. It is essential to expose and control the intact artery proximally and distally before dissection of the sac is begun. If doubt exists as to the circulation of the part, the artery can be temporarily occluded during dissection and the effect noted. Brisk bleeding from the distal end of the artery is evidence that the collateral circulation is adequate. If the circulation appears inadequate, some type of aneurysmorrhaphy or vessel graft must be considered. Suture methods directed toward repair are in general to be preferred to obliterative ligature methods, except in infected wounds, where there are diseased vessel walls, or where the vessel involved is not sufficiently vital to justify the added time and risk of restorative techniques. If possible, aneurysmorrhaphy or excision of the aneurysm with restoration of arterial continuity by means of end-to-end suture or blood-vessel graft should be carried out. If conditions are not favorable for these procedures, an attempt should be made to excise the aneurysm and suture-ligate the cut ends of the artery. It is essential to obliterate "dead space" as completely as possible. Drainage is avoided whenever possible.

## REPORT OF CASES

CASE 1.—A 43-year-old man was admitted to Harlem Hospital on April 15, 1952, complaining of a painful mass of the right thigh. He had accidentally struck this area against an automobile door on March 28, 1952. A mass appeared one week later, and it increased gradually in size.

Physical examination revealed a round pulsating firm mass, 8×8 cm. in size, just above the medial aspect of the knee. Its borders were ill-defined. The mass was moderately tender, and the overlying skin was much warmer than the corresponding skin of the other extremity. A loud rough systolic bruit was heard over the mass.

Fig. 1 (Case 1).—Arteriogram reveals a large traumatic (false) aneurysm of the superficial femoral artery.

Fig. 2 (Case 2).—Posteroanterior and lateral views of a traumatic aneurysm immediately prior to operation. The mass had been increasing gradually in size. The skin overlying the aneurysm had become ulcerated, and intermittent bleeding occurred.

Arteriography performed on April 16, 1952, revealed a large traumatic aneurysm of the superficial femoral artery (Fig. 1). On April 28th, a right lumbar sympathectomy was performed in an attempt to improve the collateral circulation prior to the direct attack on the aneurysm. On May 19th, the aneurysm was resected and a vein graft (adjacent saphenous vein) was used to bridge the defect. On May 31st, slight bleeding from the wound was noted. The oozing continued and the patient ran a low-grade febrile course. On June 22nd, there was profuse hemorrhage from the wound and the patient was taken immediately to the operating room. At operation it was found that the lower portion of the graft had given way. It was necessary to suture-ligate the femoral artery just proximal and distal to the graft. The postoperative course was uneventful, and the patient was discharged from the hospital on July 23, 1952. There was no evidence of ischemia of the involved leg at any time during the postoperative period. He has remained asymptomatic since operation.

Case 2.—A 25-year-old woman was admitted to Harlem Hospital on Oct. 14, 1952, complaining of a painful mass in the left thigh. She had been stabbed in this region on Aug. 15, 1952. At that time she was taken to the hospital and the laceration was sutured. A few days later she noted that the area just above the site of injury had become swollen and painful. Any type of motion in this area aggravated the pain. She was given penicillin and, for a time, the mass seemed to decrease in size. However, the mass then started to increase in size once again, so that at the time of admission to the hospital the overlying skin was markedly thinned out and ulcerated.

Physical examination revealed a large, tense, tender mass occupying the upper half of the inner aspect of the left thigh (Fig. 2). There were two small ulcerations of the skin overlying the mass. The left popliteal artery pulsation appeared to be normal, but the left dorsalis pedis pulse was barely palpable, and the posterior tibial pulse could not be palpated.

Arteriograms revealed a large globular aneurysm involving the middle third of the left superficial femoral artery (Fig. 3). On Nov. 10, 1952, a left lumbar sympathectomy was performed, and on Dec. 17th the aneurysm was excised and the femoral artery ligated just proximal and distal to the lesion. Since there was a large amount of "dead space" and potentially infected tissue and since the collateral circulation was excellent and the patient very young, it was deemed advisable to ligate the artery and not attempt any plastic procedures. Approximately 1200 cc. of blood was evacuated from the thigh. The

Fig. 3 (Case 2).—Arteriogram reveals a traumatic aneurysm of the superficial femoral artery.

"dead space" was obliterated as much as possible, and the skin was closed loosely with interrupted sutures. The postoperative course was uneventful, and at no time was there any evidence of ischemia of the left leg. The patient was discharged from the hospital on the 13th postoperative day, and she has remained well since that time. A bounding dorsalis pedis pulse reappeared several weeks after operation.

CASE 3.—A 17-year-old youth was admitted to Harlem Hospital on Jan. 27, 1954, complaining of a painful mass of the left arm. Two months previously he had been shot in the left elbow region, but there did not appear to be any significant injury at the time and he was sent home from the emergency ward of the hospital after the lacerations of the skin had been sutured. He had no symptoms referable to the injury until one week prior to admission, when he noted a mass just above the left elbow. The mass increased slowly in size and became painful.

Examination revealed an orange-sized tender pulsating mass just above the mesial aspect of the left elbow (Fig. 4). The overlying skin was shiny and tense. Sensation to touch and pin-prick was absent over the course of the median nerve in the hand. The left radial and ulnar pulses were not palpable, and the left hand was much colder than the right.

It was apparent that the patient had a traumatic aneurysm of the left brachial artery, just proximal to its bifurcation. The mass continued to increase in size, and operation was carried out on Feb. 2, 1954. The initial part of the procedure consisted of a stellate ganglionectomy, which was performed through a transverse incision just above the left clavicle. The stellate ganglion and the second thoracic sympathetic ganglion were excised. After this, an incision was made directly over the aneurysm and all major vessels proximal and distal to the lesion were encircled with tapes. The tapes were elevated and direct entry was made into the aneurysm. The sac was completely filled with clotted blood. When all of the clot was evacuated, a transverse jagged tear was seen in the brachial artery, 2 cm. above its bifurcation. Arterial blood spurted briskly from this opening when the adjacent tapes were released. The tear was closed over by means of several interrupted 00000 nonabsorbable surgical (silk) sutures on an Atraumatic needle. The tapes were removed; hemostasis was secured, and the wound was closed in layers.

Immediately after operation the left hand was noted to be definitely warmer than the right. The patient was ambulatory on the first day following operation, and the postoperative course was un-

Fig. 4 (Case 3).—Photograph of traumatic aneurysm of left brachial artery. The mass first appeared eight weeks after the original injury, and it increased progressively in size while under observation.

eventful. He was discharged from the hospital on Feb. 12, 1954, and has remained well since that time.

## COMMENT

The management of traumatic aneurysms is quite different from the treatment of traumatic arteriovenous fistulas. In the latter instance sudden, alarming complications are not common, and it is advisable to delay operative treatment for three to six months so that a maximum collateral circulation can develop. In the case of traumatic aneurysms, sudden complications are frequent, and it is essential to carry out operation as soon as it is feasible medically. The three patients described in this report all had typical traumatic aneurysms, and in all instances there was a period of freedom from symptoms for a significant period following the initial injury—only to be followed by sudden hemorrhage. In the first case bleeding began three weeks after trauma, and in the second and third patients sudden hemorrhage began eight weeks after the original injury. If the patients had not been admitted promptly to the hospital, serious complications might have ensued. Since these complications are so unpredictable, each patient must be examined frequently while he is awaiting surgery. If repair of arterial injuries cannot be carried out within eight hours after injury, it is advisable to delay operative treatment for approximately three to four weeks so that all hematoma and inflammation may subside and an effective collateral circulation may develop. If operation is delayed unnecessarily, there is always the possibility of sudden, alarming complications. If these complications occur shortly after injury, operation may have to be carried out as an emergency measure.

In lesions involving the lower extremity, preliminary lumbar sympathectomy is of value, since it relieves vasospasm and aids in the development of an effective collateral network. If an aneurysm is in the upper extremity, the development of ischemia postoperatively is relatively rare, and preliminary sympathectomy should be performed only if there is some doubt as to the adequacy of the collateral circulation.

At the time of operation, excision of the aneurysm and restoration of arterial continuity should be attempted. In certain instances such reconstructive procedures are not feasible and obliterative techniques are indicated. Such is the case if there is marked infection, a diseased vessel wall, or large amount of "dead space." Blood-vessel grafts should be used only if it is possible to surround them with healthy vascular tissue, since the vitality of the graft depends largely upon the ingrowth of fibroblasts from the surrounding host tissues. In Case 1 a saphenous vein graft was used to bridge a defect of the superficial femoral artery, but the graft failed because there was not sufficient healthy tissue surrounding it. When secondary hemorrhage occurred, ligation of the superficial femoral artery was performed because it was apparent at that time that the collateral circulation was adequate. In Case 2 at the time of operation it was not deemed advisable to use a vein graft because of the large amount of infected and necrotic tissue present. Since the collateral circulation was excellent, ligation of the superficial femoral artery immediately above and below the aneurysm was carried out. There was no evidence of ischemia in this extremity following operation. Several weeks after operation, bounding foot pulses became palpable. In Case 3 the adjacent tissues were healthy, and it was possible to excise the aneurysm and restore arterial continuity without compromising the size of the involved artery. Whenever possible, a procedure of this type should be carried out.

These three cases have been described in detail because each represents a distinctly different type of surgical management and yet they have all had satisfactory results. This emphasizes the fact that each patient must be considered an individual problem and that there is no one method of treatment for all cases of traumatic aneurysm.

SUMMARY

The pathogenesis, diagnosis, and management of traumatic (false) arterial aneurysms are discussed.

Three patients with typical traumatic aneurysms are described. In each instance, unexpected alarming hemorrhage occurred a considerable time after the initial injury.

In view of the hazard of undue delay, traumatic aneurysms should be operated upon as soon as all infection and hematoma have subsided and the collateral circulation appears to be adequate. During the preoperative period, it is essential to observe the patient closely in order to detect any sudden complications.

If feasible, the aneurysm should be excised and arterial continuity reestablished. However, if there is considerable infection or "dead space" or if the wall of the involved vessel is so diseased that it cannot be sutured, ligation and division of the vessel immediately proximal and distal to the aneurysm should be carried out. Before obliterative procedures are performed, the collateral circulation must be tested and found to be adequate.

Preoperative lumbar sympathectomy is a valuable adjunct in therapy, since it minimizes vasospasm and allows for the maximum development of collateral circulation.

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# Lobectomy in a Chronic Respirator Patient

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and
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## INTRODUCTION

Each epidemic of poliomyelitis leaves in its wake a number of severely disabled patients who require continued respiratory aid. It has been demonstrated in the Regional Respirator Centers established with the assistance of the National Foundation for Infantile Paralysis that most of these patients can be sufficiently improved to be placed in a home-care situation instead of continuing hospitalization with its attendant greater cost. The commonest complications encountered by these patients are minor respiratory infections and atelectasis due to the plugging of the smaller and larger bronchi with mucus which the patient, owing to his lack of an adequate cough, is unable to expel. When such an atelectasis occurs which cannot be relieved by bronchoscopic methods, exsufflation techniques, and use of detergent aerosols, chronic infection in the affected lung supervenes and the incidence of further upper respiratory complications increases in the patient. The following case is cited to demonstrate that radical surgery can be attempted in such cases to eliminate the infection and thereby further the possibility of returning the patient to a home-care situation.

## REPORT OF A CASE

A 27-year-old white farmer was well until Oct. 2, 1952, at which time he developed symptoms of malaise, lassitude, and general muscular aching, especially in the lower extremities. He continued his work in the fields four days, until he noted a temperature elevation and difficulty with breathing, which necessitated his stopping work. He consulted a physician, who confirmed a diagnosis of spinal poliomyelitis, with quadriplegic involvement. He was placed in a tank respirator, where he remained for nine months. Shortly after he was put in the respirator, a tracheotomy was performed. The patient states that he was semicomatose during the first two weeks in the hospital and has no memory of that time. Four months after the onset of his illness, while still in the tank respirator, he developed a slight cough and temperature elevation; hemoptysis occurred three times, and there was pain in the left lower chest on inspiration. A diagnosis of pneumonia was made, and the patient was treated with antibiotics. The symptoms persisted for three weeks.

The patient was admitted to our Center on July 6, 1953, while still in the tank respirator. At the time of admission his vital capacity was 600 cc.; tidal volume, 300 cc., and minute volume, 8.5 liters. X-ray of the chest on admission revealed that the entire left lung was atelectatic, with a compensatory emphysema of the right lung and with considerable displacement of the mediastinal structures into the left hemithorax. Bronchoscopy was done on July 15, 1953, at which time the left main bronchus was filled with pink purulent secretion. After bronchoscopy the patient was maintained in an atmosphere of a mucolytic aerosol therapy (Alevaire), with postural drainage, until his secretions were greatly diminished. However, repeated X-rays failed to demonstrate a significant alteration in the atelectasis. The patient was subject to many recurring respiratory infections, and, with each of these, adequate suctioning, the use of Alevaire, and the use of antibiotics resulted in temporary improvement. A second bronchoscopy was performed on Sept. 9, 1953, when he was having increased difficulties with the recurring mucous plugs.

By Sept. 28, 1953, he had been weaned completely from the tank respirator and he needed only the chest respirator and rocking bed. His unassisted time was still less than two hours per day. A continued weaning process, despite many recurring respiratory infections and excessive mucus production, enabled him to be properly ventilated with the use of the chest respirator for about 10 hours per day, the rocking bed for about 2 hours, and 12 hours

Submitted for publication May 19, 1955.

Departments of Medicine and Surgery, Creighton University School of Medicine.

From the Regional Respiratory Center, Creighton Memorial St. Joseph's Hospital, aided by the annual grant from the National Foundation for Infantile Paralysis.

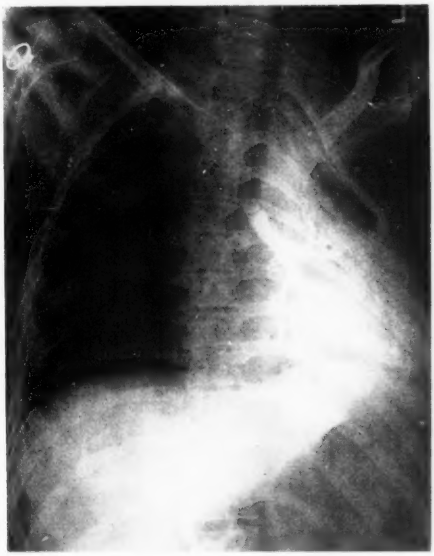


Fig. 1.—Bronchogram of left lung made on Oct. 11, 1954.

of unassisted breathing. His quadriplegia remained nearly complete, although he could be up in a chair.

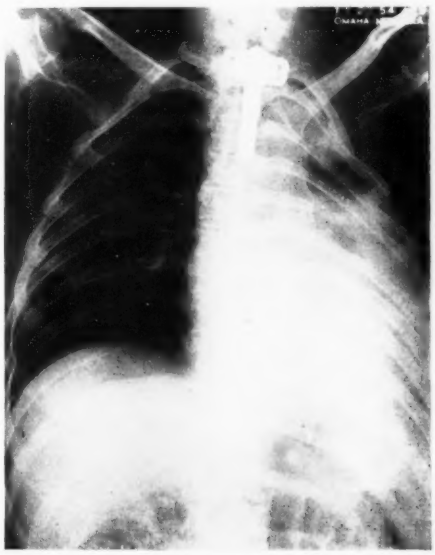
After conference, the staff decided that it would be impossible to place him in a home-care situation unless some means of preventing the recurring infection could be developed. On Oct. 11, 1954, a bronchogram was made, at which time there was demonstrated partial filling of the bronchial tree of the left lung. The left lower lobe was still markedly atelectatic, with retraction to that side. Only mild bronchiectatic changes were demonstrated in the bronchi (Fig. 1), but there were undoubtedly other bronchiectatic changes that could not be demonstrated because of retained secretions. The upper lobe of the lung could not be adequately filled for diagnostic purposes. The right lung was well ventilated. It was decided that a thoracotomy should be performed. Prior to surgery, the patient's respirometer studies were as follows: while he was lying flat after two hours with no respiratory aid, vital capacity was 1035 cc., tidal volume was 520 cc., and minute volume was 8.5 liters.

On Oct. 22, 1954, a left lower lobectomy was performed by one of us (H. H. McC.). This was done through a left posterior lateral incision, after the left fifth rib had been removed. In order to facilitate the postoperative care, it was thought best to do a second tracheotomy, since the previous one had been closed for some time. Two mushroom catheters were placed in the left hemithorax, one in the anterior fourth interspace, and one in the posterior ninth interspace. These were connected to underwater suction drainage. Throughout the operation, ventilation was maintained by an endotracheal tube with cuff, together with manual compression of the anesthetist's bag.

The report of the pathologist was as follows: "Resected left lower lobe of the lung shows bronchiectasis with chronic bronchitis, peribronchitis, atelectasis, and chronic lymphadenitis."

In the postoperative period the maintenance of adequate ventilation proved to be a difficult problem. While the tank respirator usually is the most effective means of ventilating such a patient, the presence of the catheters in the hemithorax and other tubes for intravenous medication made it difficult to secure adequate negative intratank pressure. It was then decided to use a Monaghan positive-pressure attachment through the tracheotomy tube. There was some concern as to whether this might result in a blowing out of the stump, but no ill effects occurred. The patient was therefore ventilated between Oct. 23rd and Oct. 30th by this means alone. Despite the technical difficulties encountered, the patient's $pCO_2$ and arterial oxygen saturation remained, most of the time, within normal limits. On Oct. 27, 1954, a bronchoscopy was done through the tracheotomy opening and large mucous plugs and crusts were removed. The patient's ventilation was continued with the use of positive pressure, and on Nov. 2nd, after the tubes had been removed and the dressings had been removed, he was placed in a

Fig. 2.—X-ray taken postoperatively, on Nov. 22, 1954.



chest respirator which was synchronized with the positive pressure apparatus. A new weaning program had to be instituted in a progressive manner, and by Feb. 7, 1955, the patient was using the chest respirator about two hours a day, the rocking bed about eight hours a day, and the remaining time was unassisted breathing. Spirometry at that time revealed a vital capacity of 579 cc., a minute volume of 8 liters, and a tidal volume of 307 cc. Figure 2 indicates the x-ray of the chest after surgery. The tracheotomy tube had been replaced with a plastic plug and was only occasionally used for suctioning purposes. There has been no recurrence of a major respiratory infection. On March 9, 1955, he was placed in a home-care situation in which he can be satisfactorily cared for by an attendant. The respiratory aids he requires are a rocking bed and a portable respirator for emergency needs. A program of occupational therapy compatible with his disability has been provided.

## COMMENT

As the profession of medicine becomes confronted more frequently with the dilemma of chronic illness, there is need of redefining our goals for the individual case. Physicians have the responsibility to society to consider procedures which will enable the care of the long-term seriously disabled patient to be accomplished at the lowest possible cost. The achievement of these subordinate goals involves teamwork between the various disciplines of medicine and the proper utilization of the ancillary services, such as physical therapy, occupational therapy, and medical social service.

## SUMMARY

A report of the problems involved in performing a lobectomy on a chronic respirator patient is presented.

Attention is called to the need for fairly radical procedures, if the dilemma of chronic illness is to be solved.

# Prone-Position Operating Table for Surgery of the Cardiac Coronary Sinus of Dogs

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DANIEL W. DAVIS Jr., M.D.
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During the past two years, we have been investigating the anatomy and physiology of the cardiac venous bed of dogs and have been studying the technique and value of the Beck II operation.[1]

Submitted for publication Oct. 11, 1955.

This study was aided by a grant from the New York Heart Association.

Assistant Professor of Clinical Surgery, Cornell University Medical College (Dr. Maisel); Assistant Resident Surgeon, New York Hospital, Cornell Medical Center (Dr. Davis), and Professor of Surgery and Director of the Department of Surgery, Cornell University Medical College (Dr. Glenn).

Early in our studies, it became apparent that exposure of the dorsal aspect of the heart through standard approaches was quite a difficult feat. Initially, the dog was placed in the supine position and a right or left lateral transthoracic approach to the mediastinum and heart was employed, but the exposure was not satisfactory. Particularly annoying and embarrassing were the cardiac irregularities produced by either manual or suture traction upon the heart, for these frequently resulted in ventricular fibrillation and death.

Because of these two major problems—namely, (*a*) poor exposure of the coronary sinus and (*b*) the traction which had to be exerted upon the heart—we abandoned the supine position in favor of the prone position and a left transthoracic approach. This

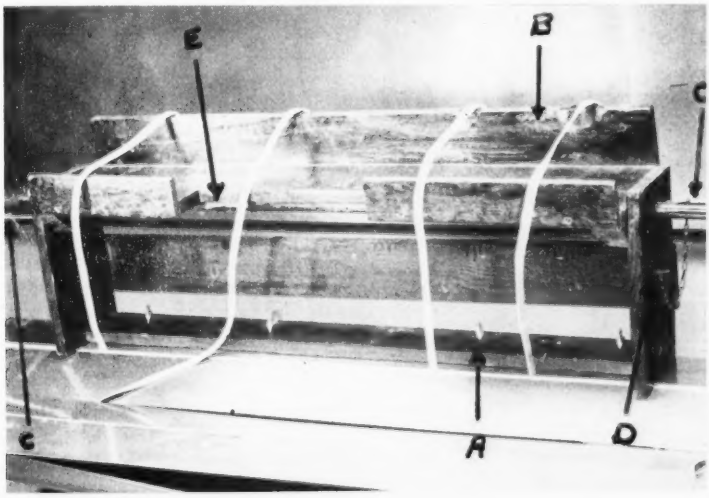



Fig. 1.—The prone-position operating table is fixed to the standard animal operating table by "C" clamps (*A*). The cradle (*B*) of this table is fixed to an axel rod (*C*) and can be rotated clockwise or counterclockwise on this rod. Position of the cradle is maintained by Dowel rods (*D*). The cutout (*E*) of this table is large enough to afford access to the left chest from anterior ribs three to seven.

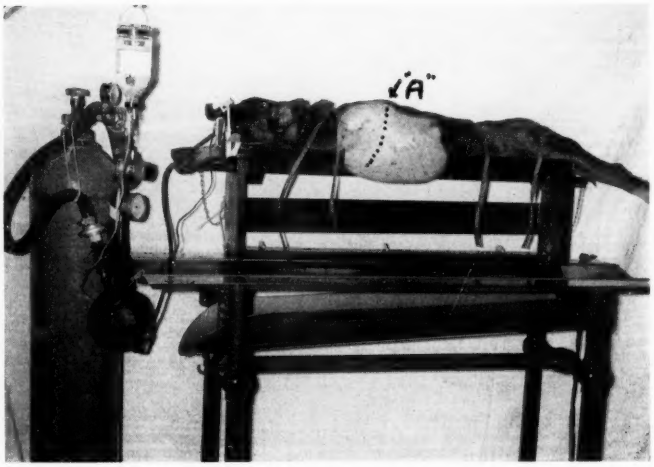
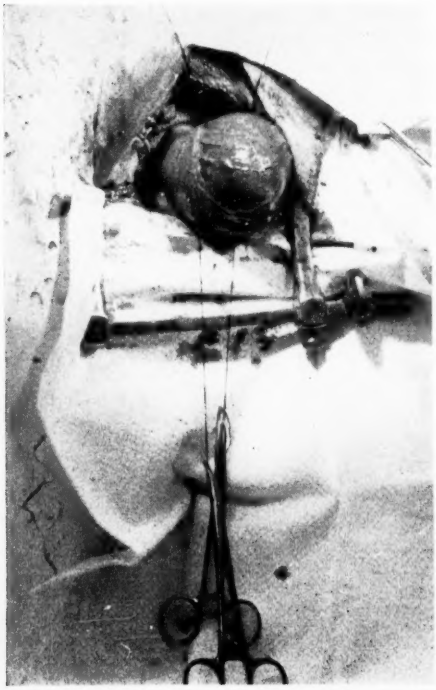



Fig. 2.—The dog is anesthetized with intravenous pentobarbital and placed in the cradle of the prone-position operating table. The chest is positioned so that the P. M. I. is in the middle of the "cutout" of the operating table. The canine trunk is comfortably and well supported in the cradle. An endotracheal tube is inserted and attached to intermittent positive-pressure oxygen apparatus. An intravenous infusion is begun in the front leg. A circumscapular incision is made from the spine to the sternum, "*A*."

Fig. 3.—The heart is exposed in the opened pericardium and then the left pleural space is made dependent by rotating the table to the left, or counterclockwise. The heart is delivered out of the chest. Gravity maintains this position of the heart. The coronary vein and sinus and the dorsal aspect of the right auricle are easily exposed. The heart is moistened with isotonic saline solution and is steadied in the cupped hand of the operator's assistant.



approach to the heart, coronary sinus, and dorsal aspect of the right auricle provided excellent exposure and obviated the need for traction upon the heart.

One of us (B. M.) then devised a prone-position operating table (Fig. 1). The procedure is as follows:

The dog is anesthetized with intravenous pentobarbital (Nembutal), shaved, and placed in the cradle of the operating table in the prone position (Fig. 2). An endotracheal tube is introduced and attached to an intermittent positive-pressure oxygen supply. An intravenous infusion is begun in the front leg. The chest is positioned so that the point of maximum impulse (P. M. I.) of the heart is in the middle of the cutout of the prone position operating table (Fig. 2). The left chest is prepared with benzalkonium chloride solution and then draped. A circumscapular incision is made from the spine to the sternum. The left thoracic cavity is entered through the intercostal space corresponding to the cardiac P. M. I. Gentle traction upon the lung exposes the mediastinum. A linear incision is made in the pericardium along the phrenic nerve, either anterior or posterior to the nerve, whichever seems more suitable. Sutures of 000 silk are then placed in the cut edges of the pericardium and attached to the drapes so that the heart is exposed in the opened pericardium and the lung tissue is retracted beneath these sutures. The left pleural space is made dependent by rotating the operating table slightly to the left, or counterclockwise, and the heart is delivered out of the chest cage by lifting it gently to the left. Gravity maintains the position of the heart in the exposed operating field (Fig. 3). The heart is kept moistened with isotonic saline solution

and is steadied in the cupped hand of the operator's assistant. The coronary vein and sinus and the dorsal aspect of the right auricle are easily exposed by incising the epicardium and dissecting away the overlying fat. After completion of the surgical procedure, the heart is gently and easily replaced into the pericardium. The pericardium is closed with interrupted 000 nonabsorbable surgical (silk) sutures, and the left lung is expanded. The table is then rotated clockwise to the right so that the dog's right chest is now dependent. Care must be taken that the intravenous infusion is not interrupted and that the endotracheal tube is not displaced during this procedure. The chest wall and skin are then closed in layers.

## COMMENT

During the past year and a half that the prone position has been employed for the exposure of the coronary sinus, we have had no operative deaths that might be attributed to fibrillation in 30 dogs. We had two deaths due to suffocation, the result of displacement of the endotracheal tube when the table was rotated. Prior to the use of this position for exposure of the dorsal aspect of the heart, we had 8 deaths in 10 dogs, as a result of fibrillation. Excellent exposure of the dorsal aspect of the heart shortens the duration of the operation, and this averts prolonged abnormal cardiac stimulations. The fact that gravity maintains the exposure of the heart during the procedure averts the need for traction sutures in the heart or manual traction upon the heart, and this greatly reduces abnormal cardiac stimulation. As a result of these two factors—(*a*) excellent exposure and (*b*) marked reduction of abnormal stimulation of the heart—we believe that the prone position and transthoracic approach to the mediastinum through the left pleura is of unusual value for exposure of the dorsal aspect of the heart.

## SUMMARY AND CONCLUSIONS

The prone position and the left transthoracic approach to the canine heart affords excellent exposure of the dorsal aspect of the heart. This operative position is particularly useful for surgery of the coronary sinus and dorsal aspect of the right auricle. Since the exposure is excellent and traction upon the heart is unnecessary, abnormal cardiac stimulations are reduced and the dangers of ventricular fibrillation lessened. An operating table for the prone position exposure of the mediastinum is described.

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# Acute Pancreatitis Following Translumbar Aortography

**Case Report with Autopsy Findings Seven Weeks Following Aortogram**

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The occurrence of untoward sequelae following translumbar aortography has been stated to be minimal in the hands of a careful operator.* It is considered by some that most accidents associated with the procedure have been caused by the use of unsuitable contrast media, such as sodium iodide.[3] Despite the fact that the newer concentrated organic iodides have given rise to fewer side-effects, a possible complication of aortography which still exists is the injection of a high concentration of dye into a major branch of the aorta. It is the purpose of this paper to report a case of acute pancreatitis following such an occurrence. This has not been reported before in the literature.

## REPORT OF A CASE

A 51-year-old white male proofreader was admitted to the Surgical A Service of Jefferson Medical College Hospital on Jan. 17, 1955. He gave a history of having enjoyed good health until four years prior to admission, at which time he began to experience a dull aching pain in both inguinal regions, radiating into the anterior and posterior aspects of both thighs. The pain was precipitated by walking and was relieved by rest. The symptoms gradually progressed in frequency and severity to a point at which the pain occasionally occurred at rest and involved the thighs, calves, and occasionally the feet. During this period weakness and fatigability of the legs was noted as well as sexual impotence. Because of these symptoms the patient was hospitalized at another institution and an aortogram was performed, revealing a thrombosis of the terminal aorta. A right lumbar sympathectomy was performed 17 months prior to admission, and, although there was some increase in the skin temperature of the right lower extremity, there was no significant symptomatic improvement.

Six months prior to admission, the patient developed paraesthesia and coldness of both lower extremities and noticed that the pain was becoming worse.

The past history and review of systems were noncontributory. Family history revealed that a maternal aunt had diabetes. The general physical examination revealed a moderately obese man. The blood pressure was 205/100 mm. of mercury in the right arm and 130/90 mm. of mercury in the left arm. The pulse was 84, and it was noted that the left brachial and radial pulsations were diminished in intensity. The temperature was 98.6 F. The eyegrounds showed Grade II arteriosclerotic changes. The pertinent findings were limited to the cardiovascular system. There was no demonstrable cardiac enlargement, but there was a Grade I systolic murmur at the apex. The femoral, popliteal, dorsalis pedis, and posterior tibial pulses were absent bilaterally, and both extremities were cold, the right being less so than the left. The skin of the legs was scaling and atrophic. The remainder of the physical examination was negative.

Laboratory studies on admission showed that the hemoglobin level was 14.9 gm. per 100 cc.; the white blood cell count was 8300 per cu. mm., and the differential count was normal. Blood serology was negative. Urinalysis showed a specific gravity of 1.016, a trace of albumin, 2+ sugar, and 1 to 3 white blood cells. Blood urea nitrogen was 20 mg. per 100 cc.; creatinine was 2.6 mg. per 100 cc., and fasting blood sugar was 131 mg. per 100 cc. The

Submitted for publication July 20, 1955.

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* References 1 and 2.

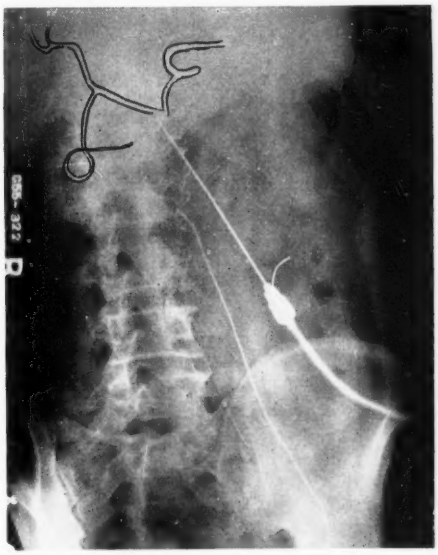


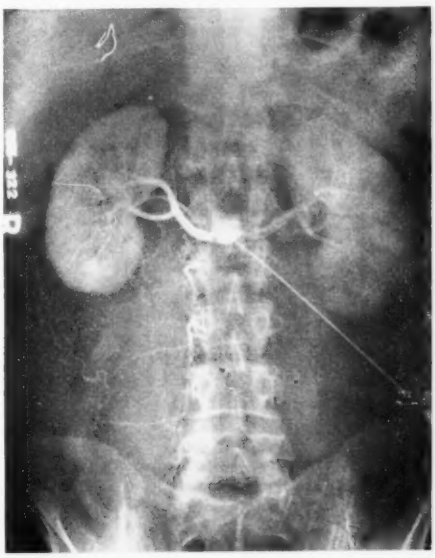
Fig. 1.—Injection of sodium acetrizoate into the celiac artery. The hepatic, splenic, and gastroduodenal arteries are visible.

glucose-tolerance curve was diabetic. Liver-function tests were within normal limits. PSP excretion was 40% in 30 minutes. Chest x-ray was normal. The electrocardiogram showed left ventricular strain.

*Hospital Course.*—In the absence of a history of diabetic symptoms, it was believed that the patient had mild diabetes, and he was accordingly treated with diet alone for the first three days in the hospital. During this period the urine sugars ranged from negative to 2+. On Jan. 21, 1955, an aortogram was performed under thiopental (Pentothal) sodium anesthesia. The standard technique for aortography was followed,[2] and 20 cc. of 70% sodium acetrizoate (Urokon sodium) was injected. The film revealed the presence of the needle in the celiac artery with opacification of its three major branches and their subdivisions (Fig. 1). After withdrawal and repositioning of the needle, another 20 cc. of sodium acetrizoate was injected. The film revealed a complete occlusion of the aorta, beginning just below the origin of the renal arteries and possibly extending into the lumen of the left renal artery (Fig. 2).

Approximately four hours after aortography, the patient was noted to appear confused and slightly cyanotic. He complained of severe periumbilical pain radiating through to his back and vomited once. There was no change in his blood pressure, and his temperature remained normal. The abdomen was distended, and there was slight epigastric tenderness. A white blood cell count done at that time was 16,000 per cu. mm. A few hours later he complained of weakness and numbness of his left leg, and examination revealed absent sensation and motor power below the left knee. During the next 48 hours, several narcotic injections were required for relief of abdominal pain. It was noted that there was increasing glycosuria, and two blood-sugar determinations performed 72 hours after the aortogram were 520 and 800 mg. per 100 cc., but there was no evidence of ketosis. It was also noted that the blood urea nitrogen had risen to 64 mg. per 100 cc. The patient was transferred to the medical service for control of his diabetes. The left leg symptoms had cleared completely at that time, and he was mentally clear. A low-grade fever was present for a few days, and leucocytosis persisted. The blood urea nitrogen returned to a normal level 10 days after the procedure. For the next six weeks of hospitalization, during which time the patient was receiving anticoagulant therapy, he required daily insulin in a dosage ranging from 64 units of regular to 40 units of isophane insulin (NPH insulin) in order to control his diabetes. On Feb. 25, 1955, an angiocardiogram was performed because of the diminished pulses in the left upper extremity, and

Fig. 2.—Injection of sodium acetrizoate into the aorta just above the renal arteries.



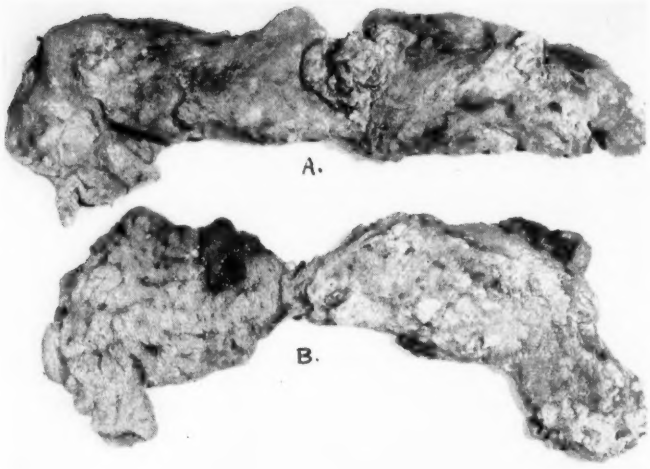



Fig. 3. — Longitudinal section of the pancreas showing outer (*A*) and cut (*B*) surfaces. Areas of softening and hemorrhage are demonstrated.

poor filling of the left subclavian vessel was noted. On March 17, 1955, a resection of the thrombosed abdominal aorta from just below the renal arteries to 1 in. below the bifurcation of the aorta, with replacement of an arterial graft, was performed by Dr. William Lemmon. At the time of surgery, extensive fat necrosis was noted throughout the abdomen. Postoperatively, the patient had good pulses in both lower extremities and appeared to be doing well until the second day, when he developed signs of a cerebrovascular accident and died.

*Autopsy Findings.*†—The body was that of a well-developed and well-nourished 51-year-old white man autopsied 20 hours after death. A recent surgical incision extended from the ensiform process to just

† The autopsy was performed by Dr. J. G. ChenSee, of the Pathology Department.

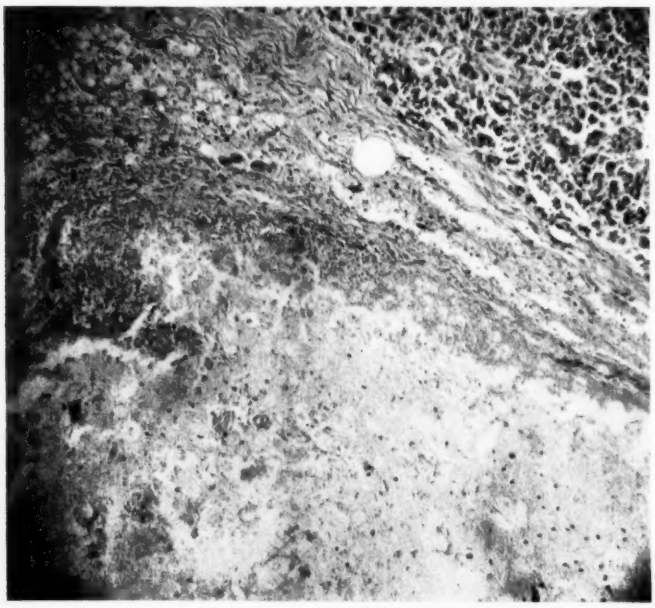


Fig. 4. — Microscopic section showing normal pancreatic tissue (upper right) adjacent to a large area of necrosis; reduced from mag. × 150.

above the symphysis pubis, over the left rectus muscle. There was focal fibrinous peritonitis in the region of the operative site. The pancreatic area contained a number of foci of fat necrosis. A recent aortic graft extended from 4 cm. inferior to the renal vessels to 2 cm. below the bifurcation of the aorta. Dissection of the aorta revealed severe diffuse arteriosclerosis with multiple foci of ulceration and calcification. A striking feature was the presence of a large fresh thrombus, which extended from the renal arterial orifices inferiorly to about 4 cm. below the anastomotic sites in the common iliac vessels. The left renal artery communicated with the aorta through a patent defect in the thrombus, but the orifice of the right renal artery proved to be occluded completely.

Attention was directed to the pancreas (Fig. 3), the vessels of which proved to be free of thrombus formation. It measured 18.5×5.5 cm. and weighed 105 gm. The pancreatic duct system was patent throughout. Greenish-yellow foci of putty-like softening were scattered over the surface of the entire organ, most extensive in the tail. These areas varied from 0.2 to 2.5 cm. in size. On section of the gland, they were found with equal prevalence through its entire substance. Microscopically, sections of the pancreas disclosed stellate expanses of necrosis about which were scattered lymphocytes and plasma cells. The process involved vascular walls traversing the necrotic tissue. Adjacent viable parenchyma contained mild diffuse interstitial lymphocytic infiltrations with some myxomatous degeneration. Examination of multiple sections failed to reveal evidence of suppuration.

## COMMENT

The immediate postaortogram symptoms and the sequence of events which followed during the next six weeks left little doubt that extensive damage to the pancreas had followed the procedure. There was also evidence of transient renal damage to what were considered normally functioning kidneys and some temporary neurological deficit in the left leg.

In reviewing the possible reasons for this patient's response to the injection, we find that the two major considerations are allergy to the dye and direct chemical trauma. Allergy seems unlikely in view of the fact that the patient had a negative skin test to sodium acetrizoate and that an angiocardiogram using 70% sodium acetrizoate was performed one month later without untoward effect. It must be postulated that this patient's pancreas, kidneys, and, possibly, spinal cord had an unusual susceptibility to chemical irritation by the organic iodide. The first injection of the dye was directly into the celiac artery, and the second injection was made adjacent to the right renal artery, resulting in a high concentration in these vessels. Because of the fact that sodium acetrizoate was the agent employed and because at the time of aortography there was a good flow of blood from the needle, a test dose of the dye was not injected in order to determine the position of the needle.

A review of the literature reveals that puncture of the right renal artery with parenchymal necrosis of the right kidney has been reported.[4] Wagner and Price reported a case of injection of sodium iodide into the superior mesenteric artery with a fatal result and one injection of sodium iodide into the celiac artery without complication.[5] Boyarsky has reported a case of possible direct chemical trauma to the spinal cord with resulting paraplegia.[6]

Rigler and Olfelt[7] reported a series of cases in which 40 cc. of sodium acetrizoate was injected into the aorta above the celiac axis for purposes of opacifying the liver and spleen. No mention was made of untoward reactions following this procedure. It should be noted that, although there was a good concentration of dye in the vessels of the celiac axis in these cases, in no instance was the needle positioned directly adjacent to the celiac artery.

In the present case the entire celiac axis was well opacified, and dye reached the pancreas through the pancreaticoduodenal artery and the pancreatic branches of the splenic artery, resulting in a diffuse necrotizing pancreatitis which exacerbated the patient's diabetes.

## SUMMARY

A case is reported in which acute necrotizing pancreatitis occurred after a translumbar aortogram. This untoward response was probably due to an excessive concentration of dye in the celiac axis. Injection of a small

amount of sodium acetrizoate to check the position of the needle would probably have avoided the complication and should be done in all aortographic procedures.

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# Rupture of Abdominal Aortic Aneurysm into the Duodenum

A Report of Two Cases

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and
WILLIAM B. CAUDELL, M.D., Cincinnati

Rupture of an aneurysm into the gastrointestinal tract is not a common occurrence. In 1942 Rottino [1] reviewed the literature and collected 32 such cases, and since that time 24 additional ones have been added.* Including this present report, the total number of cases is now 58. Since aneurysms occur principally in the aged, we will see more of this complication as our population continues to grow older. In view of this and since this disease is theoretically curable, it deserves our attention. It should be considered whenever an elderly patient with massive bleeding into the gastrointestinal tract is seen.

Aneurysms of the abdominal aorta are classified according to etiology as arteriosclerotic, syphilitic, mycotic, or traumatic.[9] Arteriosclerosis is by far the commonest cause of these aneurysms. Syphilis is the next commonest cause. Aneurysms due to other pathological processes are extremely rare. Arteriosclerosis was the cause of the abdominal aneurysm in 49 of 50 cases operated upon by DeBakey.†

The signs and symptoms of aneurysm of the abdominal aorta are as follows: (1) a pulsatile abdominal mass on physical examination and on roentgenologic examination; (2) a thrill, murmur, or bruit over the mass; (3) erosion of the lumbar vertebrae; (4) calcification in the abdominal aorta and in the wall of the mass; (5) displacement of the abdominal viscera, and (6) pain in the abdomen or back. Of all the complications of abdominal aortic aneurysm, rupture is the most dramatic and is usually the mechanism of death. Cooley [12] states that "the average period of survival after diagnosis of aneurysm of the abdominal aorta is from one to two years, with rupture ultimately occurring in most patients." With rupture the patient usually experiences severe pain and goes into shock. If the aneurysm ruptures into the gastrointestinal tract, hematemesis and melena are prominent findings. In 58 patients in which the aneurysm ruptured into the gastrointestinal tract, 23 had hematemesis or melena, 18 had abdominal pain, 12 had pain in the back, 8 suddenly collapsed, and 5 were aware of the presence of an abdominal mass.

The third portion of the duodenum is the commonest site where an aneurysm of the abdominal aorta ruptures into the gastrointestinal tract. This portion of the intestine is affected most frequently because it is relatively fixed and is in close proximity to the aorta (Fig. 1). It is therefore more likely to be damaged by this pulsating expanding abdominal lesion. Antzis [8] reported that of 49 cases of rupture into the gastrointestinal tract 36 were into the duodenum, 7 into the stomach, 5 into the jejunum, and 1 into the small bowel. Of the 36 which ruptured into the duodenum, 32 perforated into the third portion and two into the second portion. The point of perforation was unspecified in two cases.

Submitted for publication June 6, 1955.

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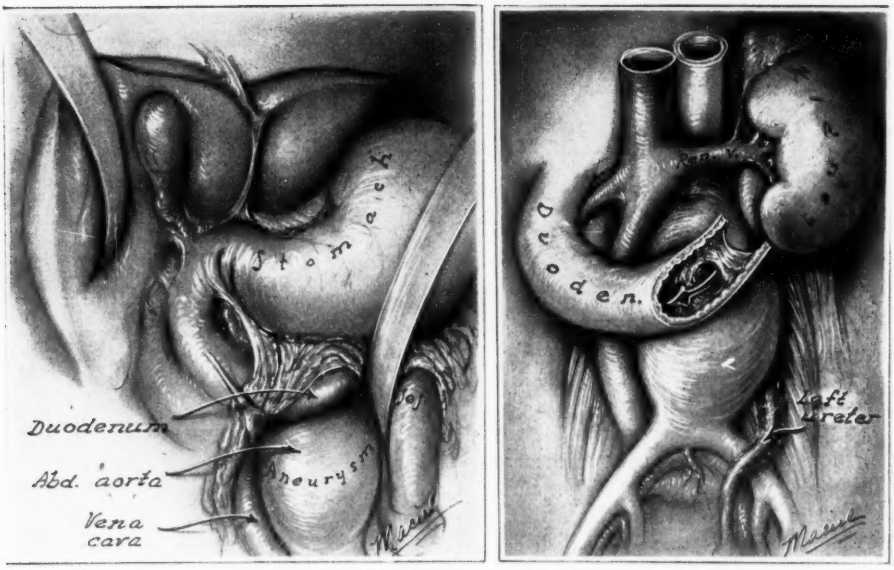


Fig. 1.—Drawing showing the close proximity of an aneurysm of the abdominal aorta to the duodenum and showing perforation into the duodenum.

REPORT OF CASES

The following two cases were recently seen at our hospital.

CASE 1.—A 59-year-old white man was admitted to the Dayton Veterans Hospital Jan. 21, 1953. For two days prior to admission he had severe burning pain of the midabdomen. On the day prior to admission the patient vomited food, old blood, and, finally, fresh blood. Three months prior to this admission he experienced similar abdominal pain, nausea, and hematemesis. He was admitted to another hospital, and his gastrointestinal tract was thoroughly examined. He was told that all studies were normal. The site of the bleeding was not determined with roentgenologic examinations of his upper and lower gastrointestinal tract. Within two days he became asymptomatic. On physical examination blood pressure was 140/90, pulse rate 96, and temperature 99.2 F. The patient was a well-developed well-nourished white man who appeared acutely ill. The anterior abdominal wall was soft, and no masses or organs were palpated. Palpation of the right and left hypochondria elicited the complaint of tenderness. The extremities appeared normal, and the peripheral pulses were of good quality.

White blood cell count was 11,000; neutrophiles, 73%; lymphocytes, 16%; monocytes, 11%; red blood cell count, 3,500,000, and hemoglobin, 10 gm. Urinalysis showed the following results: albumin, trace; sugar, 2+; 8 to 10 white blood cells per high-powered field; serum amylase, 47 units; BUN, 18 mg. per 100 cc.; prothrombin time, 68% of normal. X-ray of the chest showed tortuous calcified aorta. X-ray of the abdomen revealed irregular calcified flecks between the right iliac crest and the lower lumbar spine and considerable arteriosclerosis of the pelvic vessels.

The patient had frequent episodes of hematemesis which required 1500 cc. of whole blood and additional fluids during the initial three days of hospitalization in order to maintain his blood pressure and hemoglobin level. On the second day of hospitalization roentgenologic study of the upper gastrointestinal tract suggested the presence of an irregular constriction of the duodenal bulb. On the third day of hospitalization a preoperative diagnosis of massive hemorrhage from a duodenal ulcer was made and exploration of the abdomen was performed. A large aortic aneurysm was found, posterior to the third portion of the duodenum. The pylorus was slightly thickened and was partially adherent to the pancreas. Because the site of bleeding could not be adequately determined, the distal three-quarters of the stomach was resected. This was thought advisable because of the findings in the pylorus and because it was not apparent that the aneurysm was the source of the bleeding. On the seventh day after operation dehiscence of the wound

occurred. The wound was secondarily closed with No. 22 steel wires placed through all layers of the abdominal wall. Five hours after secondary closure the patient vomited large amounts of blood. Despite rapid transfusion of five units of blood the patient died.

Postmortem findings (Fig. 2*B*) were as follows: The duodenal closure and gastrojejunostomy were intact. The aorta at and cephalad to the origin of the patent renal arteries contained rare atherosclerotic plaques. Five perforations were found in the posterior wall of the transverse portion of the duodenum. Each measured approximately 5×7 mm. These perforations communicated with a large retroperitoneal hematoma and an aneurysmal sac 5 cm. in diameter. The defect in the anterior wall of the aorta was located where the inferior mesenteric artery normally arises. This measured 2 cm. in diameter and communicated with the aneurysmal sac previously mentioned. Microscopic examination of the aortic wall adjacent to the perforation revealed replacement with fibrous tissue. The duodenal wall at the point of perforation also showed evidence of scarring. There were zones of necrosis in the wall of the duodenum. In these zones there was old blood, a mixed chronic inflammatory-cell infiltrate, and some limited organization by fibroblasts and capillaries. A segment of aorta near the site of perforation was almost completely necrotic.

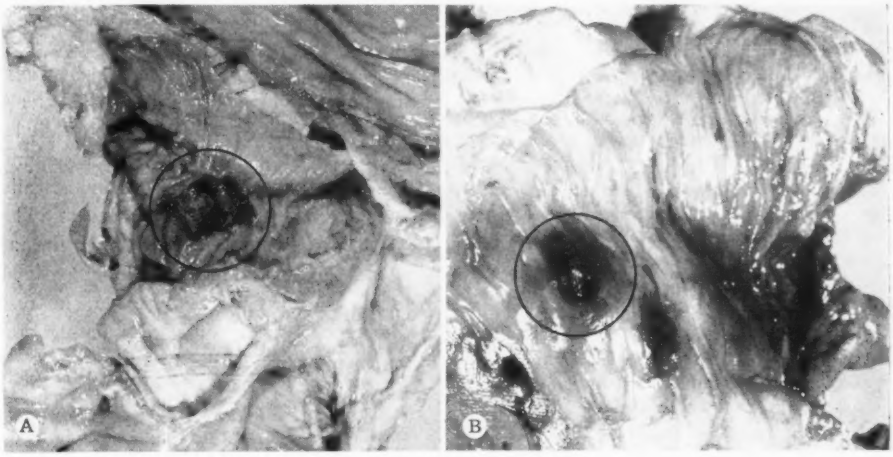


Fig. 2.—Photographs showing the defect in the posterior wall of the third portion of the duodenum which communicated with the aneurysm. *A*, Case 2; *B*, Case 1.

Case 2.—A 64-year-old white man was admitted to the Dayton Veterans Hospital March 22, 1953. Twelve hours prior to admission the patient noted the onset of severe pain in the lower abdomen. The pain radiated to the back and was unaffected by position. Nausea accompanied the pain, but the patient had not vomited. The patient was a respiratory cripple owing to pulmonary emphysema and fibrosis. Three years prior to this admission he had been forced to markedly limit his activities. He produced 2 to 3 tablespoonfuls of frothy sputum daily. There was no history of hemoptysis. On physical examination blood pressure was 80/0; pulse rate, 130, and temperature, 100 F. The patient was a well-nourished white man in a state of shock. He was dyspneic at rest. The anteroposterior diameter of the chest was greater than normal. The breath sounds were of the bronchial type. There was a slight spasm of the entire anterior abdominal wall. Peristalsis was hypoactive. No masses or organs were palpated.

White blood cell count was 16,950; neutrophiles, 95%; lymphocytes, 4%; monocytes, 1%; red blood cell count, 7,400,000; hemoglobin, 16 gm.; BUN, 26 mg. per 100 cc.; serum amylase, 105 units; serum albumin, 3.2 gm. per 100 cc.; serum globulin, 2.1 gm. per 100 cc. Urinalysis revealed the following: albumin, negative; sugar, negative; six to eight white blood cells per high-powered field; Kahn test, negative, and feces negative for occult blood. X-ray of the chest showed air under the diaphragm and x-ray of the abdomen, flat and upright, free air in the peritoneal cavity.

The diagnosis of acute perforated peptic ulcer was made, and the abdominal cavity was explored. The peptic ulcer, which had perforated through the anterior wall of the first portion of the duodenum, was found. The perforation was closed. After opera-

tion the patient had persistent spiking fever, congestive heart failure, progressive anemia, and hypoproteinemia. On the sixth postoperative day the temperature rose to 105.4 F. Blood cultures taken at this time yielded a growth of hemolytic Micrococcus pyogenes var. aureus. Treatment with erythromycin was instituted. The blood cultures produced no growth after the initiation of this therapy, and the fever subsided. The congestive heart failure responded satisfactorily to digitalis and diuretics. The patient continued to have a low-grade fever. On May 3, 1953, a pulsating mass was noted in the right upper portion of the abdomen. This was approximately 2×4 in. in size and was not tender. This mass was thought to be an aneurysm of the abdominal aorta. Multiple x-rays of the chest showed only bullous emphysema, and an x-ray examination of the upper gastrointestinal tract showed marked compression of the duodenal bulb and the antrum of the stomach by an external mass. On May 8, 1953, the patient vomited small amounts of dark-brown gastric fluid. Levin suction was instituted. On May 9, 1953, the patient went into profound shock, and blood was noted in the gastric contents. At this time it was believed that the aneurysm had ruptured into the gastrointestinal tract. The patient responded to intravenous blood and levarterenol (Levophed). He continued to vomit blood around his Levin tube and to have melena. On May 10, 1953, the patient again went into profound shock. Intravenous blood and levarterenol failed to correct his blood pressure, and he died at 11:15 a. m. on May 11, 1953.

Postmortem findings were as follows: The ulceration in the anterior wall of the first portion of the duodenum was well closed. Just to the right of the midline in the posterior wall of the third portion of the duodenum was a 3 cm. defect (Fig. 2*A*). This defect communicated with the lumen of the aorta. The rest of the gastrointestinal tract was normal. The aorta contained many atheromatous plaques. A fusiform dilatation extended from 1 cm. past the exit of the renal arteries to the bifurcation of the aorta. An anterior perforation of the dilated portion of the aorta communicated with the duodenum. Microscopic examination revealed necrosis of the media of the aorta. The wall was largely composed of hyalinized connective tissue and very thick atheromatous plaques.

## COMMENT

Rupture of an aneurysm of the abdominal aorta into the gastrointestinal tract should be included in the differential diagnosis of any case of massive upper intestinal bleeding. This is particularly true when the patient is in the older age group. When the diagnosis is made preoperatively, there is usually adequate time to prepare for surgery. The patient rarely dies with the first episode of bleeding if blood replacement is adequate. Our first patient lived for 10 days after hemorrhage, and our second lived for two days after hemorrhage. If a patient who is bleeding into the upper gastrointestinal tract is explored and the point of bleeding is not found, perforation of an aneurysm must be considered.The aorta should be palpated and an aneurysm sought. If one is found and it appears to be the source of bleeding, treatment is preferably replacement with a homograft or prosthesis.‡ If the operator is not prepared for such a contingency, it may be better judgment to close the abdomen and reoperate as soon as proper preparations can be made. Should the patient be actively bleeding from the aneurysm, it will usually be into the third portion of the duodenum. Closure of the perforation transduodenally may be a wise temporizing measure. The treatment will necessarily vary with the individual situation and require judgment and courage. With the recent increase of interest in the problem of aneurysms, some of these cases should be saved in the future.


## SUMMARY

Rupture of aneurysm of the abdominal aorta into the duodenum is discussed.

Two cases are presented.

Suggestions for treatment are made.


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# Hepatic Artery Aneurysm

## Report of a Case and Review of the Literature

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In 1809, a 50-year-old man died because of rupture of an intrahepatic aneurysm the size and shape of a heart involving the left hepatic artery. A Dr. Wilson, describing this case in a lecture before the Royal College of Surgeons, became the first to report an aneurysm of the hepatic artery.* This entity has been the subject of several excellent reviews over the past 50 years,† and a careful record of the number of cases reported has been kept. In all, 99 cases have appeared in the literature.

Admittedly something of a collector's item, this condition is nevertheless worthy of further discussion, if for no other reason than the singular lack of success surgeons have had in treating it. Among the 99 cases reported, there have been only 2 well-established "cures," and 90 patients have died of the disease. Indeed, most of the reports represent autopsies of cases in which the diagnosis was not suspected before death. Thirty-four patients have been subjected to laparotomy, and in many of these the diagnosis could not be made even at surgery.

Of the seven patients who have survived surgery, there was some doubt of the diagnosis in four. Three had intrahepatic aneurysms,‡ and for that reason the aneurysm itself was not seen. In the case reported by Anderson [2] in 1919, spontaneous hemorrhage from the right lobe of the liver was found and controlled by two mattress sutures and a gauze pack. Because the patient had previously had an aneurysm of the radial artery and subsequently had an aneurysm of the popliteal artery, it was assumed that the bleeding from the liver resulted from an aneurysm of a branch of the hepatic artery within the liver. In the case reported by Friedman [14] in 1912, the author claimed that a dilated tortuous hepatic artery was an aneurysm, despite the opinion of the surgeon to the contrary. Nothing was done at laparotomy, and the author did not state whether the pain for which the surgery was done persisted. On the basis of this case, Friedman claimed to be the first to make a preoperative diagnosis of hepatic artery aneurysm.

Three extrahepatic aneurysms have been treated surgically with survival of the patient. Perhaps the most remarkable of these was the case reported by Kehr [22] in 1903. An extrahepatic aneurysm the size of a hen's egg of the right hepatic artery was found in a 29-year-old man who had developed pain, jaundice, and hematemesis following empyema. The aneurysm, which had ruptured into the cystic duct, was treated by ligature of the common hepatic artery, packing the sac of the aneurysm, and sewing the right lobe of the liver into the wound. A 2 cm. strip of liver margin became necrotic and was trimmed off, after which the wound healed, and the patient was in good health when seen six years later. Almost 50 years afterward, Dwight and Ratcliffe [11] treated an extrahepatic aneurysm by filling the sac with wire, and, although the patient continued to have symptoms referable to the aneurysm, he survived. The only unqualified success reported since Kehr's case was the case of a 54-year-old woman reported by Quattlebaum [32] in 1954. Exploratory operation was

Submitted for publication July 8, 1955.

* Wilson, cited by Rolland.[33]

† References 11, 13, 25, 33, 34, and 47.

‡ References 2, 10, and 21.

performed because of persistent epigastric pain, and an aneurysm of the common hepatic artery was the only pathology found. The hepatic artery was ligated, and the patient was given large doses of penicillin postoperatively. She made an uneventful recovery and was completely relieved of her epigastric pain; an aortogram six months later showed no evidence of aneurysm. Thus, in only these two instances (Kehr and Quattlebaum) was an aneurysm definitely seen at surgery and treated (in both cases by ligation of the hepatic artery) with survival without further symptoms that could be attributed to the aneurysm.

### ETIOLOGY

Many causes of hepatic artery aneurysm have been given; probably in some cases the

TABLE 1.—*Etiology in Ninety-Nine Cases of Hepatic Artery Aneurysm Reported in Literature*

| | Cases, No. |
|---|---|
| Systemic infection | 33 |
| Arteriosclerosis | 18 |
| Cholelithiasis | 9 |
| Syphilis | 7 |
| Trauma | 7 |
| Cholecystitis | 4 |
| Liver abscess | 2 |
| Tuberculosis | 2 |
| Periarteritis | 2 |

associated pathology which was blamed was merely coincidental and not causative. It is interesting that infection is named so frequently. Earlier writers considered this the most important cause, although the exact pathogenesis was not given except for the statement that the infection weakened the wall of the vessel to produce the aneurysm. Among the systemic infections which were mentioned were pneumonia, osteomyelitis, bacterial endocarditis, typhoid, influenza, and mediastinitis. Stones or inflammation of the gall bladder coexisted with aneurysm frequently. In some cases the gall-bladder disease precipitated the surgery which led to the discovery of the aneurysm (and, often, the subsequent death of the patient). In two other cases described by Quattlebaum,[32] massive hemorrhage through or around the T-tube occurred during the early postoperative period after common duct exploration. In both cases the bleeding proved fatal, and autopsy showed it to be due to hepatic artery aneurysm which had not been recognized at surgery. It has been suggested that stones in the common duct by virtue of constant pressure may weaken the wall of the adjacent hepatic artery to cause an aneurysm. As might be expected in such cases, the wall of the common duct will also be weakened and the aneurysm may rupture into the duct through this area. This sequence of events should be considered when hemorrhage occurs during or after common duct exploration. External trauma, unquestionably the cause in the case reported here, has been blamed only seven times. None of these aneurysms has been called congenital.

### PATHOLOGY

Little attempt has been made to distinguish true from false aneurysms. From the descriptions that have been given, it is apparent that both have been found. The size of the aneurysms has varied from that of a "barley corn" (Rolland[33]) to that of a child's head (Wallman[45]). In 87 cases the aneurysm has been single; in 12, multiple. There have been two aneurysms in six cases; three aneurysms in three cases; four in one case; five in one case, and "several" in one case. The aneurysm was extrahepatic in 72 cases, intrahepatic in 22, and both in 3. The hepatic artery proper has been involved 47 times; the right hepatic artery, 28 times; the left hepatic artery, twice; both right and left, once; the cystic artery, four times, and the gastroduodenal artery, twice. The aneurysm has ruptured in 78 of the 99 cases. Thirty-six have ruptured into the abdominal cavity, twenty-seven into the bile ducts, seven into the gall bladder, seven into the duodenum, one into the stomach, and one into the portal vein.

### CLINICAL PICTURE

The clinical picture has usually reflected the local mechanical effects of the aneurysm, and for that reason the same symptoms and

physical signs have been present in a large percentage of patients. One would expect this consistency of findings to lead to more frequent preoperative or premortem diagnosis. This, however, has not been the case, for in only a very few has the diagnosis been suspected. Undoubtedly, this has been the result of the similarity of the symptoms of the uncommon hepatic artery aneurysm and the symptoms of the much commoner peptic ulcer and gall-bladder and common-duct disease.

On the whole, the patients have been young (average age in the 85 cases reviewed by Malloy and Jason [25] in 1942 was 38 years; however, since then none of the patients reported on has been under 40 years). Men have outnumbered women by three to one. About one-third have given a history of severe systemic infection. Previous or concurrent cholecystolithiasis or choledocholithiasis has been present in several of the cases reported recently. Pain has been the commonest symptom, having been noted in 67 instances. It has usually been epigastric or right upper quadrant pain, with much the same radiation and variation as the pain of gall-bladder disease. Only rarely has it been described as throbbing or pulsating. Jaundice, when present, usually is a manifestation of pressure of the aneurysm causing obstruction of the common bile or common hepatic duct. Although jaundice has been present in slightly less than half the cases, aneurysms of the hepatic artery proper, which would be expected to produce pressure on the common duct, amount to one-half the cases also. Thus, jaundice has been evidence that the aneurysm involved the hepatic artery proper or one of its main branches outside the liver. Massive gastrointestinal hemorrhage has been the third common symptom. This has been in the form of hematemesis, melena, or both and has been a manifestation of rupture of the aneurysm into the biliary tree, the duodenum, or the stomach. Since these aneurysms, even when extrahepatic, are not as obvious as one might expect, this entity should be considered and deliberately sought for when a laparotomy is done for bleeding and no pathology can be found in the gastrointestinal tract. This syndrome of upper abdominal pain, obstructive jaundice, and massive gastrointestinal hemorrhage is characteristic of an aneurysm of the hepatic artery. As has been mentioned, the aneurysm may rupture into the abdominal cavity, in which case the hemorrhage is usually rapidly fatal. The clinical picture of hemoperitoneum and shock developing suddenly without apparent cause has been described, and since immediate laparotomy is indicated no matter what the etiology, perhaps more of these cases can be salvaged in the future. To date, none of the 36 patients with rupture into the abdominal cavity has survived the catastrophe. Fever, mentioned frequently in earlier articles, does not occur often enough to be useful in diagnosis.

TABLE 2.—*Clinical Picture in Ninety-Nine Cases of Hepatic Artery Aneurysm Reported in Literature*

| Symptoms | Cases, No. |
|---|---|
| Pain | 67 |
| Jaundice | 42 |
| Gastrointestinal hemorrhage | 46 |
| **Location** | |
| Extrahepatic | 72 |
| Intrahepatic | 22 |
| Both | 3 |
| **Artery involved** | |
| Hepatic artery proper | 47 |
| Right | 28 |
| Left | 2 |
| Right and left | 1 |
| Cystic | 4 |
| Gastroduodenal | 2 |
| **Rupture** | |
| Peritoneal cavity | 36 |
| Bile ducts | 27 |
| Gall bladder | 7 |
| Duodenum | 7 |
| Stomach | 1 |
| Portal vein | 1 |

### TREATMENT

Many ingenious, if not very successful, methods of treating hepatic artery aneurysms have been devised. There is no doubt that treatment is necessary, as there are only two cases of spontaneous "cure" on record. Ledieu [23] in 1856 described an aneurysm the size of a hazelnut of the common hepatic

artery found on autopsy in a 25-year-old woman who died of "albuminuria and general dropsy." There were no symptoms that could be attributed to the aneurysm, and the aneurysmal cavity was quite occluded by very firm "coagulum." In 1913, Merkel § reported the case of a 56-year-old man who died 10 days after gastric resection for gastric carcinoma. An aneurysm the size of a hen's egg of the common hepatic artery was found at autopsy. The hepatic artery was entirely thrombosed and calcified, the liver having

tomy was done in the belief that the bleeding was due to peptic ulcer. In seven ¶ cholecystostomy or cholecystectomy was done; in only two of these was the diagnosis made at operation. In three cases # the common duct was explored because of persistent bleeding through a T-tube inserted at previous surgery. In all three the bleeding could be controlled with sutures or packs, but in each case fatal hemorrhage occurred shortly thereafter. In six cases the aneurysm was entered surgically, and in most of these it

TABLE 3.—*Cases with Survival Among Ninety-Nine Cases of Hepatic Artery Aneurysm Reported in Literature*

| Date | Author | Site | Treatment | Result |
|---|---|---|---|---|
| 1836 | Ledieu | Extrahepatic | None | Died of "albuminuria and general dropsy"; aneurysm found at autopsy; occluded by firm "coagulum" |
| 1903 | Kehr | Extrahepatic | Ligation of common hepatic artery and packing of sac | Liver necrosis and slough, but patient lived without further symptoms |
| 1912 | Friedman | Extrahepatic | Laparotomy only | Patient lived; surgeon felt that dilated hepatic artery was not true aneurysm; author did |
| 1913 | Merkel | Extrahepatic | None | Died 10 days after gastric resection for carcinoma of stomach; hepatic artery thrombosed |
| 1919 | Anderson | Intrahepatic | Mattress sutures and gauze pack | Patient lived; had aneurysm of radial and popliteal arteries, so spontaneous bleeding from liver assumed to be from an aneurysm |
| 1919 | Kading | Intrahepatic | Ligation of common hepatic artery | Pain, hematemesis, and melena after gunshot; Sudek ligated hepatic artery with recovery |
| 1921 | Colmers | Intrahepatic | Ligation of common hepatic artery | Patient lived; traumatic aneurysm |
| 1952 | Dwight & Ratcliffe | Extrahepatic | Wiring | Patient lived but had recurrence of melena which was a symptom of the aneurysm |
| 1954 | Quattlebaum | Extrahepatic | Ligation of common hepatic artery | Patient lived without symptoms; aortogram 6 mo. later showed no evidence of aneurysm |

been saved by collateral circulation. The aneurysm had apparently caused no symptoms.

Thirty-four patients have had the benefit of laparotomy, and, of these, twenty-seven have died during surgery or in the immediate postoperative period. In many of these 34 either the aneurysm was not recognized at surgery, or, if it was recognized, the operative procedure was not directed to the aneurysm itself. Most frequently, exploration without any definitive procedure was done. In four patients || a posterior gastrojejunos-

was packed. Death was due to either repeated hemorrhage or thrombosis of the hepatic artery and liver necrosis. In one case [34] (in which the aneurysm actually involved a branch of the hepatic artery, the gastroduodenal artery) the aneurysm was diagnosed at surgery, but because of its location in the head of the pancreas surgery directly attacking the aneurysm was considered inadvisable, and a hepaticodochojejunostomy was done to relieve the obstructive jaundice. The patient died one month later when the aneurysm ruptured. The several methods of

§ Merkel, cited by Friedenwald.[13]

|| References 8, 20, 32, and 40.

¶ References 3, 5, 17, 24, 42, and 44.

# References 29, 32, and 38.

direct attack which have been tried or suggested are listed and discussed below:

1. Ligation of the hepatic artery. This has been the most frequently used procedure and the most successful. It has been done 13 times with 4 survivors (Kehr [22] in 1903, Kading [21] in 1919, Colmers [10] in 1921, and Quattlebaum [32] in 1954). Three of these cases preceded the introduction of antibiotics by many years. Since it was not until 1949 that Markowitz, Rappaport, and Scott [26] reported on the prevention of liver necrosis following ligation of the hepatic artery by the use of antibiotics, it seems likely that survival in these cases may have been due to previous dilatation of collateral vessels or the presence of anomalous vessels which escaped ligation. Inasmuch as this is the only procedure which has met with success so far, and now that it is no longer attended with such high mortality, it will probably be more widely used in the future. Kehr,[22] in reporting his case in 1903, said, "Every case of aneurysm of the hepatic artery should be ligated, because there is collateral circulation which has developed during the formation of the aneurysm." Rolland [33] in a review on the subject in 1908 (which included a thorough tabulation of all previously reported cases) recommended surgery "with the view of ligaturing the hepatic artery, or one of its main branches, as this appears to be the only procedure which offers the patient any chance of recovery." Gordon-Taylor [15] disputed this advice, pointing out that the mortality following hepatic artery aneurysm (then 75%) exceeded the mortality following ligation for other reasons (then 60%). He cited Tandler's finding of an accessory hepatic artery in only 32% and concluded:

> Any confidence in the successful issue of a ligature of the hepatic artery must be begotten of a pious hope that some aberrant hepatic artery may supply to some degree the denuded region. . . . It is an unpalatable truth that in man, ligature of the hepatic artery or its main branches is an operation fraught with peril to life.

All surgeons will agree that ligation of the hepatic artery is far from an innocuous procedure, even with the protection of antibiotics. The hazard of liver necrosis must be weighed against the hazards of alternative procedures designed to preserve the arterial supply of the liver and still eliminate the aneurysm. When the patient's general condition is poor or when the experience of the operator is limited, the much simpler ligation is certainly indicated. Consideration should be given to the site of ligation. Studies of ligation of the hepatic and splenic arteries for cirrhosis of the liver in man [6] have shown that liver tissue is not damaged, provided the ligature is placed at the origin of these vessels from the celiac artery. Conversely, it has been stated that if all the branches of the hepatic artery are ligated as they enter the liver, the liver will not survive. The difference lies in the abundant collateral circulation provided by the gastroduodenal and the right gastric arteries. When the ligature is placed proximal to these branches, there will be a temporary lowering of pressure within the aneurysm beyond, which is the desired effect, and there will be no necrosis of the liver. However, the collateral circulation is so generous that the pressure in the hepatic artery would soon be as high as it was before operation. The alternative is given by McGregor [20]:

> Ligature of the hepatic artery proper just before it divides to enter the liver would seem, in view of the work of Tanturi, Surgart and Canepa,[39] to be justifiable and not too great a surgical hazard, provided that penicillin was administered as advised by these authors. Their dogs recovered despite the point they make, that the collateral supply to the liver is better in man than in dogs.

There will, of course, be some limitation in choice of the site of ligation imposed by the size and location of the aneurysm. However, when the aneurysm involves the main hepatic artery or one of its branches at the hilus of the liver (about 50% of cases reported), this site (just distal to the gastroduodenal and right gastric branches) appears to be the one of choice. When the aneurysm is intrahepatic, ligature of one branch of the hepatic artery at the hilus is a possibility. The appropriate branch can be determined by temporary compression to see whether the thrill or pulsation in the aneurysm disappears. According to Segall,[37] the collateral circulation

between the right and the left lobes of the liver is sufficient to prevent necrosis. Methods intended to occlude the hepatic artery gradually have been described and may prove useful in the management of aneurysm.

2. Resection of the segment of vessel bearing the aneurysm, with anastomosis of the proximal hepatic artery to the artery beyond the aneurysm. The distance between the two ends will usually be too great to bring them together.

3. Anastomosis of another artery of comparable size which can be sacrificed (e. g., the gastroduodenal or right hepatic) to the distal end of the hepatic artery after resection of the aneurysm.

4. Insertion of a vessel graft or prosthesis between the proximal and the distal ends of the hepatic artery to bridge the gap of the aneurysm.

5. Transplantation of the proximal end of the hepatic artery into the portal vein to avoid depriving the liver of its arterial blood supply. This offers a solution to the problem encountered when, after resection of the aneurysm, the distal end of the artery lies inaccessibly high in the hilus of the liver. Although successful in preventing liver necrosis in dogs, it would probably produce the complications of portal hypertension, as illustrated by Sach's case, in which rupture of an aneurysm into the portal vein was followed by fatal hemorrhage from an esophageal varix.* The technical difficulties encountered in surgery for these aneurysms make these procedures involving blood vessel anastomosis frequently impractical. Adhesions, distortion of normal anatomy, difficult exposure, proximity to vital structures, and urgency resulting from hemorrhage, shock, and poor general condition will often defeat attempts at this meticulous surgery. Although they are theoretically ideal, no clinical application of these procedures has been reported.

6. Wrapping the aneurysm with cellophane and dicetyl phosphate to produce a firm fibrous envelope about the aneurysm.† This has been used for aneurysms of the aorta with limited success in relieving symptoms and preventing extension of the aneurysm. However, it would not be applicable in many cases of hepatic artery aneurysm, namely, those that are intrahepatic and those that have produced biliary obstruction.

7. Wiring. Insertion of lengths of flexible wire into the aneurysm with or without subsequent electrothermic coagulation to produce gradual occlusion of the lumen of the aneurysmal sac while maintaining flow of blood at the center where the greater rate of flow prevents clotting has been described and used successfully by Blakemore [7] in treating aneurysms of the aorta. Dwight and Ratcliffe [11] stated in their report on a case of hepatic artery aneurysm:

> A Blakemore needle was inserted into the aneurysm, and .009 stainless steel wire, which had been previously roughened with emery cloth, was pumped in until the aneurysm would hold no more. A few more feet of wire were inserted at a different point. In all, 31 feet of wire were used. Both times removal of the needle was followed by brisk hemorrhage, which ceased in a few minutes when the wire was cut off, and Gelfoam soaked in thrombin held with pressure over the puncture site.

However, as mentioned before, their patient continued to have melena, and they felt that further surgery might be necessary. This procedure is attractive because it is not technically difficult and does not require the hazardous dissection necessary in other procedures.

8. Aneurysmorrhaphy. The methods of obliterating aneurysms described by Matas [27] are effective and are as applicable to hepatic artery aneurysms as to others. Control of hemorrhage by temporary compression of the artery proximal to the aneurysm is necessary in all cases. In the case of a sacculated aneurysm the sac is opened, the clots are removed, and the opening between the vessel and the sac is closed with sutures and reinforced by suturing the walls of the sac over it. Matas called this procedure restorative endoaneurysmorrhaphy. When a fusi-

* Sach, cited by Dwight and Ratcliffe.[11]

† References 4, 19, 31, and 48.

form aneurysm is found, the sac is similarly opened and the clots are removed. Then, both arterial orifices and all branch openings are closed from within by individual transfixion ligatures, and the sac is obliterated by successive rows of sutures. This procedure was called obliterative endoaneurysmorrhaphy and, of course, had the effect of ligating the artery. When preservation of the artery is necessary or desirable, as in the case of the hepatic artery, Matas advocated reconstructive endoaneurysmorrhaphy:

> The continuity of the parent artery may be restored by making a new channel out of the sac-walls, which can be brought together by sutures over a guide (catheter or drainage tube) inserted into the proximal and distal openings of the aneurysm. Before the last suture is tied, the guide is removed, and the channel is left behind, corresponding to the outline of the original artery.

The latter method was used by Gordon-Taylor [15] in 1943 in treating an aneurysm of the common hepatic artery in a 61-year-old woman. The aneurysm had ruptured into the gall bladder to produce hematemesis and melena. After dissecting the gall bladder from the liver, he opened the gall bladder and identified a small opening in the mesial aspect of the wall by which it connected with the aneurysm. Hemorrhage from the opening continued despite efforts to compress the hepatic artery. The gall bladder was removed, and a considerable "button" of aneurysmal wall was left on the artery and a reconstructive aneurysmorrhaphy performed, the remains of the sac being sutured in layers. Although the patient died 40 hours after operation (Gordon-Taylor felt that the infection present, Streptococcus and Escherichia coli having been cultured from blood taken from the wall of the aneurysm, precluded success in this case), the artery was found to be patent at autopsy. The author thought that the difficulties of aneurysmorrhaphy were not as great as those of ligation. The method used in the case presented below was obliterative endoaneurysmorrhaphy.

## REPORT OF CASE

A 27-year-old white man entered Cook County Hospital March 20, 1954, because of hematemesis. Past history revealed that he had been shot in the abdomen eight months before, in August, 1953. The bullet had entered 1 cm. below the right costal margin at the midclavicular line and lodged beneath the skin directly posterior to this point, just below the middle of the 12th rib. He had been taken to another hospital, where he remained for 10 days and was treated with gastric suction, fluids, and blood transfusion. He remembered seeing blood in the gastric tube aspirate the first day. No laparotomy was performed. The bullet was removed from beneath the skin.

He had considered himself well until about one week before admission, when he awoke with nausea and vomiting. The vomitus contained streaks of blood. The symptoms subsided until three days before admission, when he began to have cramping epigastric pain that radiated to the right costal margin. The day before admission he vomited blood, passed dark red blood per rectum, and collapsed.

Physical examination on admission revealed a well-developed well-nourished man who appeared pale but was in no acute distress. Blood pressure was 120/70; pulse rate, 92, and temperature, 99.2 F (rectum). Heart and lungs were normal. The abdomen was soft and nontender. There was a small round scar below the right costal margin at the site of the entrance of the bullet. No masses or organs were palpable. Bowel sounds were normal, and there was no murmur or bruit. Rectal examination revealed dark red blood on the examining finger.

A Levin tube was inserted into the stomach, and a small amount of red blood was aspirated. Urinalysis revealed 1+ bile and 1+ urobilinogen. Hematocrit reading was 35%. Blood volume was 5326 cc., with an estimated deficit of 1234 cc.

Management was similar to that in a case of bleeding ulcer, with gastric suction, absolute bed rest, sedation, and administration of atropine, penicillin, and blood transfusions. Although no more blood returned through the Levin tube, it was apparent that he had continued to bleed, for the hematocrit reading was still 35% after 5 pt. (about 2500 cc.) of blood had been given. When, after 24 hours, the bleeding did stop, he appeared quite healthy and was without symptoms. About one week later, while still in the hospital, he had another episode of massive hemorrhage necessitating further transfusions. The bleeding stopped promptly when the above measures were reinstituted.

Blood Kahn test was negative. NPN was 34 mg. per 100 cc. Total protein was 6.7 gm. per 100 cc. Icteric index was 25. Alkaline phosphatase was 5.6 units. Cephalin flocculation was 0 units. Thymol turbidity was 3.8 units. Gamma globulin was 1.01 gm. per 100 cc. Chest x-ray showed normal heart and lungs. Barium enema revealed a normal colon. On two attempts at cholecystography the gall bladder could not be visualized even with a double

Barium meal x-rays taken before surgery, showing the concave defect of the first portion of the duodenum caused by pressure of the aneurysm.

dose of dye. Barium meal revealed a persistent, smooth, concave defect on the greater curvature of the duodenal bulb. Gastric analysis revealed 104 units of free acid after histamine. Proctoscopy disclosed no abnormalities.

Diagnosis considered at this time, before surgery, were duodenal ulcer and gall-bladder disease. An aneurysm of the hepatic artery was strongly suspected because of the history of trauma, massive gastrointestinal bleeding, pain, jaundice, and the x-ray evidence of a mass just above the duodenum.

With the patient under cyclopropane-ether anesthesia, the abdomen was opened through a right subcostal incision extended later into the right flank. A distended gall bladder surrounded by adherent omentum was found. The gall-bladder area was explored, and the dissection was carried down to the region of the common duct. Just below the gall bladder (between it and the hilus of the liver) lay a thick-walled pulsating aneurysm about 7 cm. in diameter. It closely resembled a dilated Hartmann's pouch in position and appearance, but needle aspiration revealed bright red blood. To the right of the aneurysm and contiguous with it was a thin-walled encapsulated hematoma filled with degenerating blood. The aneurysm could not be separated from its bed in the liver. The foramen of Winslow, obliterated by adhesions, was opened enough to allow the admission of one finger. While the encapsulated hematoma was being dissected away

from the aneurysm, the latter was entered. The vigorous bleeding which followed could not be controlled with hemostats; therefore, the wound was packed with gauze. The patient's blood pressure had fallen to shock level. No further surgery was done during the next 30 minutes, during which time he was given blood until his blood pressure returned to normal and more blood for transfusion was made available in the operating room. As soon as the packing was removed, a noncrushing Statinsky clamp was placed across the portal triad by inserting one blade into the foramen of Winslow. The bleeding stopped. The gall bladder was opened and was found to contain old clots of blood. A fistula between the lumen of the gall bladder and that of the aneurysm was found posteromedially in the wall of the gall bladder. The opening was partially occluded by clots at the time of surgery but was easily reopened by blunt dissection and admitted a finger tip. The incision in the gall bladder was carried down across this opening, and the aneurysmal sac was opened widely. After removing blood and clots from the sac and with the suction tip in the field, the Statinsky clamp across the portal triad was partially released and reapplied. By noting the location of bleeding in the aneurysm, the two openings of an artery were identified in the posterosuperior wall of the sac. The artery was about 0.5 cm. in diameter and by its course was identified as the right hepatic artery. It appeared that the superior wall of the vessel was intact, but there was a gap of 1.5 cm. in the lower wall that communicated with the aneurysm. Arterial blood pulsed from the opening at each end when the Statinsky clamp was released. This was convincing evidence that there was good collateral circulation to the right lobe of the liver beyond the aneurysm. Therefore, both openings were sutured closed with fine silk. When the Statinsky clamp was released now, there was no bleeding. The gall bladder and some of the excess aneurysmal sac were removed, and the rest of the sac wall was folded in to occlude the lumen. The common duct was identified. The cystic duct and artery, which had been stretched across the wall of the aneurysm, were individually ligated. The left hepatic artery could be felt pulsating in the hilus of the liver. A Chaffin tube was inserted in the right gutter and brought out through a stab wound. The fascia and peritoneum were closed as one layer with interrupted No. 32 stainless steel wire sutures. The skin was closed with silk.

While recovering from anesthesia, the patient vomited a large amount of blood and clots. (Bleeding into the gall bladder had undoubtedly occurred during manipulation, and the blood had passed via the cystic and common ducts to the duodenum and stomach.) He aspirated some of the vomitus and became cyanotic. Breath sounds were absent over the entire right chest until the tracheobronchial tree was thoroughly aspirated of blood and clots. His color promptly improved.

His postoperative course was remarkably smooth and uneventful. He received 100,000 units of aqueous penicillin every three hours and 600,000 units of procaine penicillin twice daily. The Chaffin tube was removed on the seventh day after surgery. There was no further bleeding, jaundice, or pain.

## SUMMARY

Previously reported cases of hepatic artery aneurysm are discussed. Ninety-nine cases have appeared in the medical literature. Only two aneurysms seen at surgery and successfully treated have been reported.

The clinical picture is described, and the methods of treatment are listed: (*a*) ligation of the hepatic artery, (*b*) resection and anastomosis, (*c*) anastomosis of another artery to the hepatic artery, (*d*) vessel graft, (*e*) transplantation of the hepatic artery into the portal vein, (*f*) wrapping, (*g*) wiring, and (*h*) endoaneurysmorrhaphy.

An aneurysm of the right hepatic artery with a fistula into the gall bladder and massive bleeding into the gastrointestinal tract is reported. The correct diagnosis was strongly suspected before surgery. The reasons for this diagnosis were (*a*) history of gunshot wound of the abdomen, (*b*) pain in the right upper quadrant, (*c*) hematemesis and melena, (*d*) jaundice, (*e*) nonvisualization of gall bladder, and (*f*) extrinsic defect of the duodenum on barium meal.

The aneurysm was successfully treated by obliterative endoaneurysmorrhaphy. The fundamental steps in the procedure used in this case were (*a*) temporary occlusion of the hepatic artery proper by a noncrushing clamp across the portal triad; (*b*) boldly opening the aneurysm widely; this made the identification of the openings in the hepatic artery possible without prolonged and dangerous dissection in the hilus of the liver; (*c*) closure of these openings in the artery from within the aneurysm; this had the effect of ligating the right hepatic artery, and large doses of penicillin were therefore given postoperatively; this may have been superfluous, however, since there was evidence of good collateral circulation.

It is felt that the methods of endoaneurysmorrhaphy described by Matas offer the safest and most certain means available for the treatment of aneurysm of the hepatic artery.

1360 E. 48th St. (15).

ADDENDUM

Since the preparation of this paper, three more cases of hepatic artery aneurysm have been reported, two of which were successfully treated by cellophane wrapping (Steelquist, J. H.: Aneurysm of the Hepatic Artery, *Am. J. Surg.* **89**:1241, 1955).

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# Congenital Cystic Dilatation of the Common Bile Duct

**Report of Three Cases, Analysis of Fifty-Seven Cases, and Review of the Literature**

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**and**
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Congenital cystic dilatation of the common bile duct is a very important, albeit a very uncommon, condition. Only 242 cases have been reported in the literature since Vater [76] published the first case report, in the year 1723. The total number includes 175 cases reviewed by Shallow and his associates in 1943,[63] 57 cases reported since then and analyzed in this review, and 10 cases reported individually * for which data were not available for inclusion here.

The present study undertakes to bring the literature up to date and to report three new cases in detail. Although many different designations have been used for this disease, the term "congenital cystic dilatation of the common bile duct" is the one regarded as most appropriate.

## REPORT OF CASES

CASE 1.—A 27-year-old woman was admitted in 1948 to the gastroenterological service of the Whittington Hospital (London, England) as an emergency, with the diagnosis of cholecystitis. She complained of upper abdominal pain, colicky in nature and radiating through to the back. At the age of 15 years she had had an attack of jaundice accompanied by violent upper abdominal colic. Four weeks prior to the present admission she had had a similar attack that was less severe and with no apparent jaundice.

On examination the patient obviously was in distress, rolling with pain. There was guarding in the right upper quadrant of the abdomen, with a positive Murphy sign. The temperature was 99.8F; pulse rate, 100, and respiratory rate, 20 a minute. Pertinent laboratory findings included positive tests for bile in the urine. Hemoglobin was 10.2 gm. per 100 cc., and white blood cell count, 4400/cu. mm. The gall bladder could not be visualized by oral cholecystography.

The pain subsided with conservative management. At laparotomy, performed on March 27, 1948, the gall bladder was found to be normal. In the free edge of the gastrohepatic omentum there was a cystic dilatation of the common bile duct of about "the size of a tangerine," pushing the gall bladder up and the duodenum down. There were large blood vessels coursing across its surface. No other abnormality was present. The cystic dilatation was anastomosed by a two-layer anastomosis to the first part of the duodenum (Figs. 1, 2, and 3). A drainage tube was brought down to the site of anastomosis, and then the abdomen was closed. Recovery was uneventful, and the patient was discharged two weeks after surgery.

Six months later there was a recurrence of attacks of epigastric tightness of variable severity, occasionally accompanied by vomiting. Jaundice never was present. Findings on the physical examination, excepting epigastric tenderness, were normal. Laboratory data were within normal limits. A cholecystogram showed no visualization of the biliary tract. Results of barium-meal examination showed a small persistent fleck of barium near the first and second parts of the duodenum, which was presumed to be the site of the anastomosis. Since barium did not pass into the cystic dilatation, it was concluded that the patient probably had a

Submitted for publication May 14, 1955.

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* References 6, 8, 14, 27, 57, 47, 51, 61, 67, and 73.

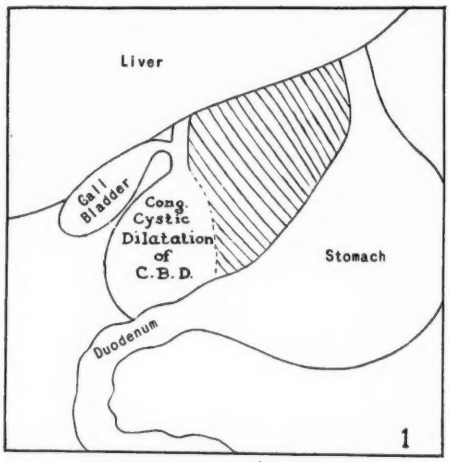



Fig. 1 (Case 1).—Diagram of preoperative appearance of congenital cystic dilatation of the common bile duct.

stricture of the anastomosis. The symptoms persisted, and a second operation was performed on Oct. 15, 1951, three and one-half years after the initial procedure. The anastomosis was found to be stenosed (Fig. 3); there was a small stone and some organic matter above the stricture (Fig. 4). The cystic dilatation was dissected free from the duodenum; the strictured anastomosis was separated (Fig. 5*A*), and an end-to-end anastomosis to the jejunum of the Roux-en-Y type was performed (Fig. 5*B*). The patient recovered and was discharged two weeks after operation with a normal serum bilirubin level. The patient reported 30 months after the second operation that she felt perfectly normal.

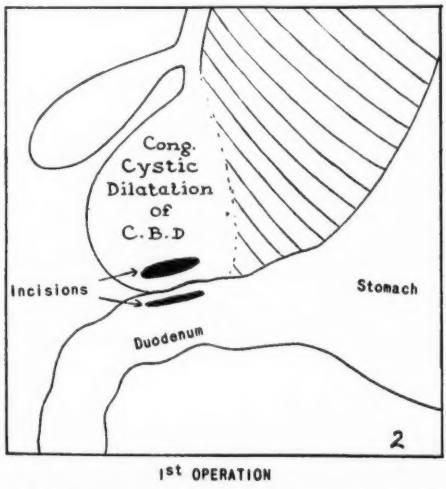



Fig. 2 (Case 1).—Diagram of anastomotic incisions for choledochoduodenostomy.

Case 2.—A 34-year-old woman was admitted to the Cleveland Clinic Hospital on Jan. 23, 1954, with a year's history of intermittent midepigastric pain. The pain during attacks had been severe and continuous, frequently radiating around both costal margins, and occasionally extending directly through to the back. The pain often was associated with nausea, vomiting, and chills, but never with jaundice or fever.

Previously, at operation on Dec. 3, 1953, in another hospital, the patient was found to have a severe suppurative pancreatitis. There were no stones in the gall bladder, and the common bile duct was not visualized. A drain was placed into the lesser omentum bursa, but improvement was not satisfactory.

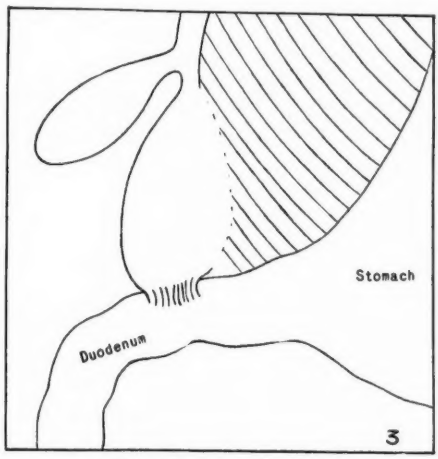



Fig. 3 (Case 1).—Diagram of appearance after choledochoduodenostomy.

Physical examination upon admission to the Cleveland Clinic Hospital was essentially negative, except for some residual tenderness in the epigastrium. There was no palpable mass and no jaundice.

Laboratory studies were within normal limits, except for a serum amylase level of 2100 Myers and Killian units per 100 mg. Barium examination of the esophagus, stomach, and duodenum gave results within normal limits. The gall bladder did not fill on three separate oral cholecystograms. During conservative management, several acute

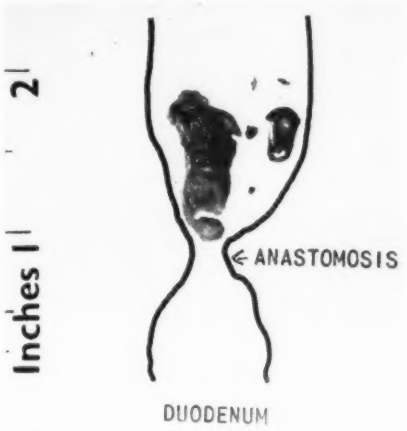



Fig. 4 (Case 1).—Diagram of postoperative stricture of anastomosis; photograph of calculi found in common duct at second operation.

attacks of abdominal pain occurred with levels of serum amylase ranging to 2020 units per 100 mg.

A diagnosis of biliary tract disease with chronic recurrent pancreatitis was made, and on Feb. 4, 1954, operation was performed. A cystic dilatation of the common bile duct, 6 cm. in diameter, was identified and mobilized. Needle aspiration of the cyst revealed bile. An operative cholangiogram (Fig. 6) demonstrated the passage of the dye from the cyst proximally into the hepatic radicles and distally into the duodenum. The cyst was opened, revealing numerous small crystals and multiple openings that communicated with the proximal common bile duct and cystic duct. There was also a common opening of the pancreatic and common bile ducts with a fistula of the common bile duct. Transverse incision was made in the duodenum immediately opposite the ampulla of Vater, and probes were passed without difficulty from the cystic dilatation through the ampulla. A sphincterotomy was performed, and a T-tube was introduced through the cystic dilatation, the proximal limb extending into the proximal duct and the distal limb extending through the ampulla of Vater into the duodenum. The major portion of the cystic dilatation was resected so as to allow a double-layer closure over the T-tube. This repair was reinforced with adjacent fibers and fatty tissue. The operation was concluded with a cholecystectomy, and a Penrose drain was placed in the foramen of Winslow. Convalescence was uneventful, and the patient was discharged 10 days after operation.

Five months later, the patient began to experience intermittent colicky pain in the right upper quadrant which radiated to the back and the right scapula. The pain was accompanied by nausea and vomiting. Since the end of August, the patient had had icteric sclerae and several acholic stools.

On readmission to the Cleveland Clinic Hospital, on Sept. 9, 1954, the only positive physical finding was that of icterus. Results of laboratory studies were within normal limits, except that the serum bilirubin was as follows: direct-reacting, 78 mg. per 100 ml. and indirect-reacting, 40 mg. per 100 ml.; the urine was positive for urobilinogen. Cholangiographic studies were negative.

A tentative diagnosis was made of choledocholithiasis or stricture of the distal common bile duct. At operation, on Sept. 10, 1954, the common bile duct was found to be dilated to a diameter of 5 cm.

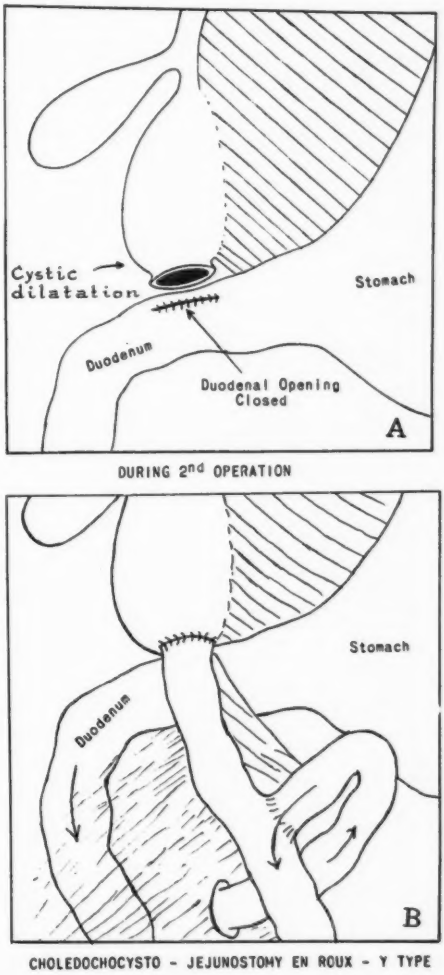



Fig. 5.—*A*, during second operation; *B*, choledochocystojejunostomy, Roux-en-Y type.

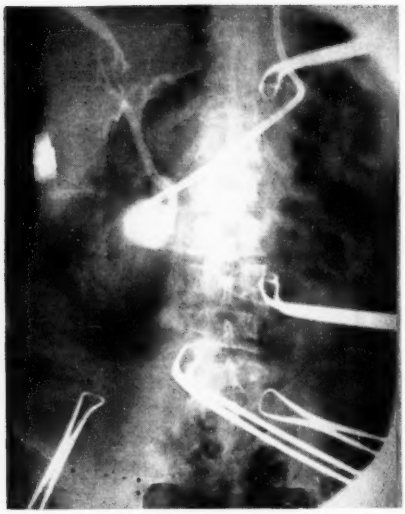
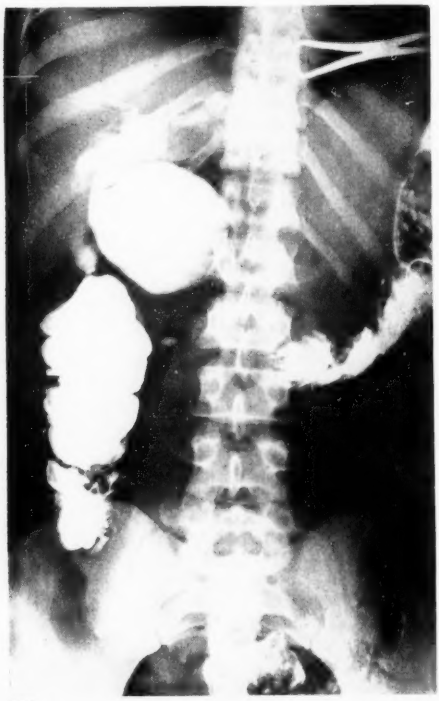


Fig. 6 (Case 2).—Cholangiogram at operation, showing congenital cystic dilatation of the common bile duct.

Fig. 7 (Case 3).—Cholangiogram at operation, showing congenital cystic dilatation of the common bile duct.



and aspiration returned murky bile and several stones. A 2 cm. choledochotomy revealed thickening of the duct wall to 4 mm. and narrowing of the distal portion of the common bile duct. A side-to-side choledochoduodenostomy was effected. The patient had an uneventful convalescence and was discharged 17 days after operation.

Case 3.—A 58-year-old white salesman was seen at the Cleveland Clinic Hospital on Sept. 25, 1951, with a four-day history of right upper quadrant abdominal pain and gradually developing jaundice. The pain was associated with vomiting, chills, and fever. Stools were light-colored, and the urine was dark. Previous health had been good, except for flatulence following the ingestion of beans, cabbage, and greasy food.

Physical examination revealed an acutely ill moderately jaundiced elderly man. Temperature was 104 F, and pulse rate, 124 beats a minute. There was marked tenderness with muscle guarding in the right upper quadrant, where a vague mass was noted.

Pertinent laboratory findings were positive tests for bile and urobilinogen in the urine, a serum amylase level of 1281 Myers and Killian units per 100 mg., a serum alkaline phosphatase level of 1.8 Bodansky units, and a cephalin-cholesterol flocculation test that was negative in 48 hours. The hemoglobin was 14.8 gm. per 100 cc., and the white blood cell count, 9600 per cu. mm.

Roentgen-ray examination disclosed a large incidental hiatus hernia and nonfunctioning gall bladder (double-dose technique). A presumptive diagnosis of gallstones with acute cholecystitis and acute pancreatitis was made.

At operation, on the day after admission, the gall bladder was found to be distended, thick walled, and acutely inflamed, but it contained no stones. A cholangiogram (Fig. 7) showed a circumscribed pocket of the opaque medium 3 to 5 cm. in diameter at the distal end of the common bile duct, interpreted as a cyst. Exploration of the common duct showed the cyst to contain only bile. There were no calculi. Probes were passed with some difficulty into the common bile duct, probably because of induration and thickening of the head of the pancreas. Cholecystectomy was performed, and a side-to-side two-layer anastomosis between the cystic dilatation and the adjacent duodenum was performed. Biopsy specimens were taken of the pancreas and the cyst, confirming a chronic pancreatitis. A T-tube was left in the common bile duct.

Convalescence was uneventful. When last seen, two years and 11 months after the operation, the patient was asymptomatic.

## REVIEW OF PERTINENT FACTORS

The rarity of congenital cystic dilatation of the common bile duct is shown by repre-

TABLE 1.—*Sex Distribution*

| Sex | Cases, No. | | Cases, per Cent | |
|---|---|---|---|---|
| | 57 (This Study) | 232 (Total) | 57 (This Study) | 232 (Total) |
| Females | 37 | 172 | 65 | 74.1 |
| Males | 20 | 60 | 35 | 25.9 |
| Totals | 57 | 232 | 100 | 100 |

sentative statistics: In 17,381 operations of the biliary system performed at the Mayo Clinic before 1926, only 2 cases were reported [33]; in 757,000 admissions to the Presbyterian Hospital (New York) there were only 2 cases.[66] In the Whittington Hospital (London, England), only 1 case was found in 26,520 admissions (Case 1, this report). At the Cleveland Clinic Hospital 2 cases (Cases 2 and 3, this report) were the only cases in 192,507 hospital admissions from 1923 to 1954. In the 232 cases in which sufficient data are available for analysis, the condition appears more frequently in females (74.1%) than in males (25.9%) (Table 1). Of the 57 cases herein analyzed, more than 80% are described in persons less than 30 years of age (Table 2), although this condition may be found at any age.

*Etiology.*—The etiology of this condition has been the subject of much speculation and remains obscure. One group of writers regards it as resulting from the action of extrinsic or acquired factors. The majority of writers, however, favor congenital origin as the most logical explanation of the condition. Supporting this view are the following facts: 1. The condition is found in children and the newborn, whereas acquired types of biliary tract disease are unusual before the age of 30. 2. The condition is found associated with congenital anomalies of other organs. 3. In the adult cases, there usually is no other obvious explanation to be found such as a regional inflammatory reaction, obstruction distal to the cystic dilatation, or a history of trauma.

The specific embryologic abnormality which could produce cystic dilatation of the common bile duct has been reported by a number of authors.[5] They include malformation of the common bile duct †; narrowing of the common bile duct from below by persistence of fetal physiologic epithelial occlusion ‡; narrowing of the intramural portion of the duct [20]; angular insertion or abnormal course of the duct into the duodenum, which causes kinking and obstruction §; diverticulum from the common bile duct ||; presence of a valve-like fold of tissue over the mouth of the intraduodenal portion of the duct [56]; rests of pancreatic tissue in the wall of the duct,

TABLE 2.—*Distribution of Fifty-Seven Newly Analyzed Cases of Disease According to Patients' Age Group and Sex*

| Age of Patient when Disease Was Diagnosed | Patients, No. | Sex of Patient | | Total Cases, per Cent | |
|---|---|---|---|---|---|
| | | Male | Female | | |
| 1-11 mo | 8 | 3 | 5 | 14.1 | 82.6% less than 30 yr. of age |
| 1- 4 yr | 9 | 2 | 7 | 15.8 | |
| 5- 9 yr | 4 | 3 | 1 | 7.0 | |
| 10-14 yr | 5 | 2 | 3 | 8.7 | |
| 15-19 yr | 7 | 2 | 5 | 12.3 | |
| 20-24 yr | 6 | 2 | 4 | 10.5 | |
| 25-29 yr | 8 | 3 | 5 | 14.0 | |
| 30-39 yr | 3 | 1 | 2 | 5.2 | 17.4% more than 30 yr. of age |
| 40-49 yr | .. | .. | .. | .... | |
| 50-59 yr | 3 | 2 | 1 | 5.2 | |
| 60-69 yr | 3 | .. | 3 | 5.2 | |
| 70-79+ yr | 1 | .. | 1 | 1.8 | |
| Totals | 57 | 20 | 37 | 100 | |

† References 39 and 62.

‡ Bohm, F., cited by Shallow, Eger, and Wagner.[63]

§ References 3 and 60.

|| Winternitz, cited by Shallow, Eger, and Wagner.[63]

which weaken it and permit local dilatation [11]; accessory gall-bladder formation from the common bile duct [11]; postnatal infection or abdominal trauma [84]; weakness of the wall of the duct,¶ and achalasia of the sphincter of Oddi.[79]

The most plausible theory, based on sound embryologic principles, is that which attributes the condition to congenital malformation of the common bile duct.# According to Yotuyanagi,[83] the etiology lies in the inequality of proliferation of the epithelial cells of the proximal portion of the common bile duct at the stage when the primitive common bile duct still is solid and at the stage of physiologic epithelial occlusion. There may occur energetic proliferation of the cells of the upper segment during the stage of physiologic epithelial occlusion and a less energetic proliferation of the lower portion during the same stage so that, as recanalization occurs, the upper part of the duct will be abnormally wide and the lower part relatively narrow.

*Pathology.*—The pathology of the disease has been discussed in detail by many authors.*

Characteristically, the cystic dilatation of the common bile duct occurs 2 cm. above the duodenum. The wall of the cystic dilatation may range in thickness from 2 to 10 mm. Histologically, the wall is lined with cylindrical or cuboidal epithelium that usually is covered by irreguar deposits of bile pigments and supported by connective tissue, occasionally elastic fibers, and smooth-muscle elements. In rare instances mucous glands are found. Purulent exudate or ulcerations of the inner surface may be present. Valve-like folds, 1 to 2 cm. in diameter, have been described by some authors.† When present, they are adjacent to the openings into the ducts. The content of the cystic dilatation is bile, the character of which depends on the amount of obstruction and on the status of the liver function. The cystic dilatation varies in size from that of a small cherry to a mass distending the entire abdomen.‡ Apart from the case of Swartley and Weeder,[72] in which there was a double common bile duct with a cystic dilatation of the right duct, other interesting findings have included a communication between the cystic dilatation and the pancreatic duct,[28] cystic dilatation associated with accessory hepatic ducts,[44] cystic dilatation suggesting a re-formed gall bladder,[53] cystic dilatation containing stones and undergoing cancerous change,[3] coexistent primary hepatic carcinoma,[2] cystic dilatation associated with congenital atresia of the bile duct,[58] obstruction of the distal end of the common bile duct by the cystic dilatation with low level of serum bilirubin (2.5 mg. direct-reacting and 5.1 mg./100 ml. indirect-reacting), and cystic dilatation with free communication with both cystic and pancreatic ducts (Case 2, this report).

Commonly accompanying findings are hepatic cirrhosis and cholangitis.§

*Clinical Features.*—"Pain, jaundice, and tumor" constitute the classic triad, and when all three occur together in a young girl the condition of congenital cystic dilatation of the common bile duct is highly probable.

The occurrence of the triad is by no means constant in these cases (Table 3). In our review, the triad was present in 33 (57.7%) of 57 and in 147 (63.3%) of 232 cases. The pain (63.4% of 232 cases) usually is localized in the right upper quadrant and is cramping in nature. The jaundice (69% of 232 cases) is oftener than not intermittent. The tumor (72% of 232 cases) has the greatest clinical significance, typically is rounded, soft to the touch, and movable laterally but not vertically. The mass occasionally is found toward the left side of the abdomen [11] or, if very large, may occupy the entire abdomen. Symptoms produced, however, are not necessarily related to the size of the cystic mass. Anorexia, vomiting, diarrhea or constipation, and anemia may accompany this condition. Enlargement of liver

¶ References 7 and 41.
# References 42 and 83.
* References 11, 15, 54, 64, 78, and 83.
† References 49 and 60.
‡ References 19, 24, 37, 38, 43, 44, 56, 63-66, 71, 74, and 83.
§ References 9, 17, 18, 24, 26, 34, 80.

TABLE 3.—*The Diagnostic Triad in 57 Newly Analyzed Cases and in 232 Total Cases*

| Triad of Symptoms | Data for 57 Cases* | | | Data for 232 Total Cases† | | |
|---|---|---|---|---|---|---|
| | Present, Cases, No.‡ | Absent, Cases, No. | Questionable or Not Reported, Cases, No. | Present, Cases, No.‡ | Absent, Cases, No. | Questionable or Not Reported, Cases, No. |
| Pain | 44 (77.1) | 6 (10.6) | 7 (12.3) | 147 (63.4) | 67 (28.9) | 18 (7.7) |
| Jaundice | 38 (66.7) | 19 (33.3) | .. | 160 (69) | 68 (29.0) | 4 (2.0) |
| Tumor | 33 (57.8) | 19 (33.3) | 5 (9.9) | 167 (72) | 56 (24.6) | 9 (3.4) |

* Triad of symptoms was present in 33 of 57 instances (57.7%).
† Triad of symptoms was present in 147 of 232 instances (63.3%).
‡ Figures in parentheses are percentages.

and spleen may occur, and Banti's syndrome has been described in advanced cases.

The clinical picture may vary from a complete absence of symptoms to the full-blown bile peritonitis, usually fatal, which follows a rupture of the cystic dilatation from trauma, necrosis, or inflammation.‖ Additional features related to cirrhosis of the liver (such as ascites), or to secondary infection (such as abscess of the liver), are described. Eighteen cases have been detected during pregnancy.¶

*Diagnosis.*—The diagnosis may be suspected from the clinical investigation of the patient on the basis of the signs and symptoms already referred to but is made with finality only at operation. Neugebauer, in 1924,[49] was the first to diagnose such a condition preoperatively. The difficulty is proved by the high rate of error in the preoperative diagnosis in 232 collected cases: diagnosis correct, 36 cases (15.5%); diagnosis incorrect, 196 cases (84.5%) (Table 4).

Roentgenography should be of benefit, but cholecytography seldom has revealed the lesion, because the cystic dilatation cannot concentrate the opaque matter. New intravenous dyes currently used to demonstrate the common bile duct may be of aid in visualizing the cystic dilatation. Urwitz[75] succeeded in ascertaining preoperatively the existence of a cystic dilatation with calcification of the wall of the cystic dilatation, by injection of a contrast medium directly into the cystic dilatation through the abdominal wall. The same result has been achieved by others.# A plain roentgenogram of the ab-

‖ References 4, 5, and 82.
¶ References 13 and 21.
# References 14, 37, and 69.

TABLE 4.—*Preoperative Diagnoses in 57 Newly Analyzed Cases and in 232 Total Cases*

| Preoperative Diagnosis | In 57 Cases | | In 232 Cases | |
|---|---|---|---|---|
| | No. | Per Cent | No. | Per Cent |
| Correct | | | | |
| Choledochus cyst* | | | | |
| A. First diagnostic impression | 8 | 14 | 23 | 9.9 |
| B. Mentioned as possible or suspected | 6 | 10.5 | 13 | 5.6 |
| Total correct diagnoses | 14 | 24.6 | 36 | 15.5 |
| Incorrect | | | | |
| Cholecystitis, cholelithiasis, or choledocholithiasis | 11 | 18.9 | 32 | 14.2 |
| Pancreatic cyst or tumor | .. | .... | 12 | 5.1 |
| Retroperitoneal cyst or tumor | 4 | 7 | 10 | 4.3 |
| Echinococcus cyst | 1 | 1.7 | 18 | 7.7 |
| Cyst of liver | 2 | 3.5 | 5 | 2.1 |
| Hydrops or enlarged gall bladder | 1 | 1.7 | 5 | 2.1 |
| Obstructive jaundice or cirrhosis of liver | 4 | 7 | 7 | 3 |
| Miscellaneous | 17 | 29.6 | 52 | 22.4 |
| No diagnosis or none stated | 3 | 5.2 | 55 | 23.7 |
| Total incorrect diagnoses | 43 | 75.4 | 196 | 84.5 |

* Constitutes diagnosis of "congenital cystic dilatation of the common bile duct."

TABLE 5.—*Types of Treatment in Fifty-Seven Newly Analyzed Cases*

| Types of Treatment | Cases, No. | Recoveries, No. | Deaths, No. | Mortality Rate, % |
|---|---|---|---|---|
| Total | 57 | 39 | 18 | 31.5 |
| No surgical treatment | 5 | 0 | 5 | 100 |
| Surgical treatment | 52 | 40 | 12 | 23 |
| Drainage and/or marsupialization | 6 | 1 | 5 | 83.3 |
| Choledochocystogastrostomy | 2 | 1 | 1 | 50 |
| Choledochocystopylorostomy | 1 | 1 | 0 | 0 |
| Choledochocystoduodenostomy | 20 | 17 | 3 | 15.0 |
| 1. Marsupialization, drainage, and secondary choledochocystoduodenostomy | 5 | 5 | 0 | 0 |
| 2. Primary choledochocystoduodenostomy and/or cholecystectomy | 15 | 12 | 3 | 20 |
| Excision of congenital cystic dilatation | 11 | 8 | 3 | 27 |
| 1. Choledocho- or hepatico- or hepaticocholedochoduodenostomy | 3 | 2 | 1 | 33.3 |
| 2. Excision, longitudinally, of outer wall of congenital cystic dilatation and reconstruction of wall into a common bile duct | 2 | 2 | 0 | 0 |
| 3. Excision of congenital cystic dilatation and gall bladder, choledochocystojejunostomy, and jejunojejunostomy | 1 | 1 | 0 | 0 |
| 4. Partial excision of anterolateral wall of congenital cystic dilatation (longitudinally) and choledochogastrostomy | 1 | 1 | 0 | 0 |
| 5. Removal of 9/10 of congenital cystic dilatation and primary anastomosis of dome of congenital cystic dilatation to duodenum | 1 | 1 | 0 | 0 |
| 6. External drainage of congenital cystic dilatation and gall bladder; secondary excision of congenital cystic dilatation, closure of fistula from gall bladder | 1 | 1 | 0 | 0 |
| 7. Partial excision of congenital cystic dilatation, external drainage of common bile duct and hepatic ducts, and secondary anastomosis of gall bladder to upper end of common bile duct; choledochoduodenostomy | 1 | 0 | 1 | 100 |
| 8. Excision of congenital cystic dilatation; no procedure* | 1 | 0 | 1 | 100 |
| Anastomosis, Roux-en-Y type | 5 | 5 | 0 | 0 |
| 1. Primary choledochocystojejunostomy, Roux-en-Y type | 3 | 3 | 0 | 0 |
| 2. Cholecystectomy, drainage, and secondary choledochocystojejunostomy, Roux-en-Y type | 1 | 1 | 0 | 0 |
| 3. Choledochocystoduodenostomy and secondary choledochocystojejunostomy, Roux-en-Y type | 1 | 1 | 0 | 0 |
| Choledochojejunostomy | 2 | 2 | 0 | 0 |
| Endocholedochorrhaphy | 1 | 1 | 0 | 0 |
| Cholecystectomy and Vitzel's operation | 1 | 0 | 1 | 100 |
| Cholecystectomy only (true condition of common bile duct not having been detected) | 3 | 3 | 0 | 0 |

* This case proved to be inoperable: atresia of hepatic and common bile ducts.

domen may indicate the presence of a cystic tumor. A roentgenogram of the stomach will show any displacement of the pylorus and first part of the duodenum by the cystic dilatation and even the cystic dilatation may be clearly seen. McLaughlin [43] considers that the existence of air in the cystic dilatation is pathognomonic of the condition. Wright [82] suggests additional radiologic signs that may lead to the diagnosis, such as calcification of the cystic wall, a comma-shaped gall bladder outlined by dye high above the duodenum, and sometimes a slight dilatation of the right renal pelvis in the intravenous pyelogram. Echinococcus cyst of liver and omentum, pancreatic cyst, hydrops of the gall bladder, mesenteric cyst, and retroperitoneal tumors are the chief diseases to be considered in the differential diagnosis of a cystic mass in the same general location as the cystic dilatation of the common bile duct. The easiest means of establishing the diagnosis in the operating room is by operative cholangiography, as demonstarted in our Cases 2 and 3 (Figs. 6 and 7).

*Treatment and Prognosis.*—The outlook in the past for congenital cystic dilatation of the common bile duct, judging by the reported cases, has been very bleak. The overall mortality in 148 cases treated by operation was 51%.[63] It is clear that newer operative techniques together with a more precise knowledge of the condition will improve the results, and a much more accurate picture is to be obtained by analyzing results in the 57 cases reviewed in this paper. Here 52 of 57 patients were operated upon, with a mortality rate of 23% (Table 5).

TABLE 6.—*Types of Treatment in 232 Cases of Congenital Cystic Dilatation of the Common Bile Duct*

| Types of Treatment | Cases, No. | Recoveries, No. | Deaths, No. | Mortality Rate, % |
|---|---|---|---|---|
| No surgical treatment | 30 | 1* | 29 | 96.6 |
| Surgical treatment | | | | |
| Aspiration only or marsupialization; drainage ± cholecystectomy | 52 | 18 | 34 | 65.5 |
| Choledochocystoduodenostomy | 91 | 67 | 24 | 26.3 |
| 1. Marsupialization; drainage and secondary choledochocystoduodenostomy | 28 | 21 | 7 | 25 |
| 2. Primary choledochocystoduodenostomy ± cholecystectomy | 62 | 46 | 16 | 25.8 |
| 3. Choledochocystoduodenostomy through elastic tube | 1 | 0 | 1 | 100 |
| Excision of congenital cystic dilatation | 35 | 18 | 17 | 48.5 |
| 1. Excision of congenital cystic dilatation and choledocho- or hepatico- or hepaticocholedochoduodenostomy | 10 | 6 | 4 | 40.0 |
| 2. Excision of congenital cystic dilatation with primary anastomosis of ends of duct | 1 | 1 | 0 | 0 |
| 3. Excision of congenital cystic dilatation ± gall bladder and choledochojejunostomy ± jejunojejunostomy | 3 | 3 | 0 | 0 |
| 4. Excision of the anterior wall of congenital cystic dilatation (longitudinally) and reconstruction of wall into a common bile duct | 2 | 2 | 0 | 0 |
| 5. Partial excision of anterolateral wall of congenital cystic dilatation (longitudinally) and choledochogastrectomy | 1 | 1 | 0 | 0 |
| 6. Excision of congenital cystic dilatation with drainage and secondary anastomosis to intestine | 1 | 1 | 0 | 0 |
| 7. Excision of congenital cystic dilatation, secondary cholecystenterostomy | 1 | 0 | 1 | 100 |
| 8. Excision or attempted excision of wall of congenital cystic dilatation ± drainage | 12 | 2 | 10 | 83.3 |
| 9. External drainage of congenital cystic dilatation and gall bladder and secondary excision of congenital cystic dilatation; closure of fistula from gall bladder | 1 | 1 | 0 | 0 |
| 10. Partial excision of congenital cystic dilatation; external drainage of common and hepatic ducts and secondary anastomosis of gall bladder to upper end of common bile duct; cholecystoduodenostomy | 1 | 0 | 1 | 100 |
| 11. Removal of 9/10 of congenital cystic dilatation and anastomosis of congenital cystic dilatation to duodenum | 1 | 1 | 0 | 0 |
| 12. Excision of congenital cystic dilatation (case inoperable; coexistent atresia of hepatic and common bile ducts) | 1 | 0 | 1 | 100 |
| Choledochocystojejunostomy, Roux-en-Y type | 5 | 5 | 0 | 0 |
| 1. Primary choledochocystojejunostomy, Roux-en-Y type | 3 | 3 | 0 | 0 |
| 2. Cholecystectomy, drainage, and secondary choledochocystojejunostomy, Roux-en-Y type | 1 | 1 | 0 | 0 |
| 3. Choledochocystoduodenostomy and secondary choledochocystojejunostomy, Roux-en-Y type | 1 | 1 | 0 | 0 |
| Choledochocystogastrostomy | 3 | 2 | 1 | 33.3 |
| Choledochocystopylorostomy | 1 | 1 | 0 | 0 |
| Marsupialization ± subsequent aspiration ± gastroenterostomy | 4 | 0 | 4 | 100 |
| Laparotomy, secondary cholecystoduodenostomy, subsequent aspiration and drainage of congenital cystic dilatation, subsequent laparotomy, and drainage | 1 | 1 | 0 | 0 |
| Cholecystenterostomy | 4 | 4 | 0 | 0 |
| Endocholedorrhaphy | 1 | 1 | 0 | 0 |
| Nature of operation not known | 5* | 0 | 4 | 80 |
| Total | 232 | 118 | 113 | 48.7 |
| Totals of cases surgically treated | 202 | 117 | 84 | 41.5 |

* The result of one case is not known.

The multiplicity of procedures employed is clearly shown in Table 6. This reflects the uncertainty among surgeons in dealing with the condition. It is futile to attempt to analyze mortality by specific procedure, but it is worth noting that the present-day opinion clearly points to the importance of type of anastomosis between the cystic dilatation and the intestinal tract, in one stage or in two. The anastomosis may be a side-to-side choledochocystoduodenostomy, as first described by Bakes,* in 1907, or a choledochocystojejunostomy of the Roux-en-Y type, introduced by Keeley [35] in 1948. To avert regurgitation of intestinal contents into the common bile duct, the length of the bowel between the choledochocystojejunostomy and the enteroenterostomy should exceed 12 in.†

* Cited by Shallow, Eger, and Wagner.[63]

† References 16, 52 and 69.

Morbidity and mortality should in the future be greatly reduced by two principal factors: (1) a clearer understanding of the existence of the entity of the congenital cystic dilatation of the common bile duct so that the diagnosis may be suspected beforehand but certainly may be made accurately at operation; (2) an understanding of the principle of sidetracking the cyst to duodenum or jejunum at the time of surgery.

Morbidity and mortality will, of course, continue to be affected by factors which affect any operation: the condition of the patient at the time of operation and the character of the preoperative and postoperative care. However, the crux of the matter is the preoperative recognition of the disease (Table 7).

TABLE 7.—*Correlation of Preoperative Diagnosis and Mortality Rate*

| Status of Preoperative Diagnosis | Cases, No. | Recoveries, No. | Deaths, No. | Mortality Rate, % |
|---|---|---|---|---|
| In 57 analyzed cases (this study) | | | | |
| Correct or suspected | 14 | 11 | 2 + 1* | 21.4 |
| Incorrect or none made | 42 | 28 | 15 | 31.6 |
| In total 232 cases† | | | | |
| Correct or suspected | 36 | 25 | 11 | 30.5 |
| Incorrect or none made | 195 + 1† | 85 | 110 | 56.6 |

* Inoperable case; atresia of hepatic and common bile ducts.
† The result in this case is not known.

### SUMMARY OF 57 NEWLY ANALYZED CASES

The 57 cases collected since the report of Shallow and his associates [63] include the 3 cases presented in this study and are summarized in Table 8. The trends shown in surgical treatment and the improved outlook already have been mentioned.

### DISCUSSION OF TERMINOLOGY

A glance at the references will make it clear that we are confronted with a variety of expressions for the single clinical entity under discussion. The following terms illustrate the existing confusion in terminology: Congenital choledochus cyst ‡; congenital cystic dilatation of the common bile duct §; congenital cyst of the common bile duct ||; idiopathic dilatation of the common bile duct ¶; congenital malformation of the bile duct [24]; choledochus cyst #; megalocholedochus or megacholedochus *; megahepatocholedochus [55]; congenital diverticulum [26]; congenital dilatation of the hepatocholedochus.[68] Because of a lack of a single definite term recognized by all, medical writers in preparing treatises interchange most of the above terms.

Most of the above terms may be excluded, as they are not sufficiently consistent with present-day basic concepts. The term "cyst of the common bile duct" or "choledochus cyst" defines a cyst somewhere along the bile duct, but the condition of which we write is a total cystic dilatation of the duct. The term "cystic dilatation of the common bile duct," while describing the present condition, does not suggest the origin of this lesion. The terms "megalocholedochus" or "megacholedochus" are not applicable. In Greek, the word is μεγαλοχοληδόχος and comprises two component words: μεγάλος and χοληδόχος. Μέγας or μεγάλος, meaning large, big, are used specifically to denote enlargement,†

‡ References 25, 41, 75.

§ References 31, 35, 45, 50, 54, and 63.

|| References 5, 32, and 78.

¶ References 36, 39, and 83.

# References 1, 4, 13, 17, 43, and 72.

* References 10, 61, 68, 77, and 79.

† There are many similar Greek terms characterizing conditions of varying enlargement, e. g., μεγαλοσπληνία — megalosplenia, μεγαλοκαρδία — megalocardia, μεγαλογλωσσία — megaloglossia, μεγαλοκύτταρα — megalocytes, etc., an analysis of which reveals an enlargement, or augmentation, but never a cystic dilatation or any condition related to cysts.

TABLE 8.—*Congenital Cystic Dilatation of the Common Bile Duct: Fifty-Seven Cases Not Analyzed in Previous Reviews*

| No. | Author | Year of Publication | Sex | Age, Yr. | Symptoms | | | Preoperative Diagnosis | Treatment | Results and Follow-Up |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Pain | Jaundice | Tumor | | | |
| 176 | Smith [66] (1st case) | 1942 | F | 20 | + | + | + | Pancreatic, hepatic, choledochal, or mesenteric cyst | Marsupialization and subsequent choledochocystoduodenostomy | Well, 11 mo. postoperatively |
| 177 | (2d case) [66] | 1942 | F | 28 | + | + | + | Cholelithiasis | Excision of cystic dilatation, drainage, and secondary hepaticocholedochoduodenostomy and cholecystectomy | Died on 10th postoperative day |
| | Shallow, Eger, & Wagner [63] (1st case)* | 1943 | M | 20 | + | + | + | Choledochus cyst with obstructive jaundice | Excision of cystic dilatation; cholecystectomy and hepaticoduodenostomy | Well, 3 yr. and 8 mo. postoperatively |
| 178 | (2d case) [64] | 1946 | F | 58 | — | + | + | Choledochus cyst | Choledochocystoduodenostomy | Well, 7 mo. postoperatively |
| 179 | Gray [26] | 1942 | F | 39 | + | — | — | Gallstone colic | Choledochocystoduodenostomy | Recurrent symptoms of cholangitis |
| 180 | Diebold [19] | 1942 | M | 5 | + | — | + | Retroperitoneal tumor | Excision of the cystic dilatation and hepatoduodenostomy | Recovery |
| 181 | Wollesen [81] (1st case) † | 1942 | F | 63 | + | + | — | Cholecystitis | Cholecystectomy | Recovery |
| | | 1942 | F | 66 | + | + | — | Cholecystitis | Cholecystectomy | Recovery |
| 182 | (2d case) † [81] | 1942 | F | 76 | + | + | — | Cholecystitis | Cholecystectomy | Recovery |
| 183 | (3d case) † [81] | | | | | | | | | |
| 184 | Swartley [71] | 1943 | F | 22 | + | + | + | Hydrops of the gall bladder | Cholecystectomy and choledochoduodenostomy | Recovery, but died 1 yr. postoperatively of abscess of liver |
| | Weeder [89] (2d case)* | 1939 | M | 14 | .. | .. | .. | ..... | ..... | Dec. 13, 1932, excision of cystic dilatation and right hepatic duct; 1936, splenomegaly, splenectomy (during operation, atrophy of right lobe and compensatory hypertrophy of left lobe of the liver were noticed); 1942, thrombocytopenia, fatal; gastrointestinal hemorrhage from esophageal varices owing to portal obstruction; at autopsy, cirrhosis of liver with esophageal varices |
| 185 | Hutchins & Mansdorfer [31] | 1944 | F | 6 | + | — | + | Neuroblastoma | Cholecystostomy, choledochocystoduodenostomy | Recovery |
| 186 | Irwin & Morison [32] | 1944 | M | 30 | + | — | — | Appendicitis | Appendectomy; choleecystogastrostomy; drainage of congenital cystic dilatation | Died on 1st postoperative day |
| 187 | Misra & Singh [48] | 1944 | M | 8 | + | + | + | Amebic hepatitis | Aspiration; marsupialization | Died on 21st postoperative day |
| 188 | Chesterman [13] | 1944 | F | 29 | + | — | + | Final clinical diagnosis: choledochus cyst | Aspiration; choledochocystoduodenostomy | Recovery |
| 189 | Michel [46] (1st case) | 1944 | F | 12½ | + | — | + | Tumor of right upper quadrant of abdomen | 1. Aspiration, marsupialization and drainage; 2. (after 4½ mo.) enteroeystostomy | Recovery |
| 190 | (2d case) [46] | .... | F | 1 | + | — | ? | Perforated appendix | Aspiration; marsupialization | Died on 18th postoperative day |

TABLE 8.—*Congenital Cystic Dilatation of the Common Bile Duct: Fifty-Seven Cases Not Analyzed in Previous Reviews*—Continued

| No. | Author | Year of Publication | Sex | Age, Yr. | Symptoms | | | Preoperative Diagnosis | Treatment | Results and Follow-Up |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Pain | Jaundice | Tumor | | | |
| 191 | Turner [74] (1st case) | 1944 | F | 4 | + | — | + | Probable appendiceal abscess | 1st operation: ext. drainage of congenital cystic dilatation and cholecystostomy; 2d operation: (after 6½ mo.) excision of congenital cystic dilatation | Recovery |
| 192 | Turner [74] (2d case) | 1944 | M | 3 ♀ mo. | + | — | + | Intussusception | Marsupialization | Died, 24 hours postoperatively |
| 193 | Brachetto-Brian, Garre, & Dominicis [9] | 1945 | F | 27 | + | + | + | Choledochus syndrome | 1. Choledochostomy: aspiration, drainage of congenital cystic dilatation (after two years); 2. marsupialization-drainage | Died first postoperative day |
| 194 | Bravo & Munoz [10] | 1945 | F | 15 | + | + | + | Hydatid cyst of the liver | Marsupialization and drainage of the congenital cystic dilatation; choledochocystogastrostomy | Operative death |
| 195 | Gaertner [22] | 1946 | M | 14 | + | — | + | Hepatic or mesenteric cyst | Endocholedorrhaphy (reconstruction of the congenital cystic dilatation according to the technique of Matas and as used for aneurysms) | Recovery |
| 196 | Blegen & Boyer [4] | 1946 | F | 17 | + | + | + | Rupture of gall bladder or, probably, of choledochus cyst | 1. Incision and drainage of biliary abscess; 2. after 2 mo. and 13 days, partial excision of cystic dilatation; external drainage of common and hepatic ducts and anastomosis of ampulla of gall bladder to upper end of common bile duct; cholecystoduodenostomy through a T-tube | Died on 18th postoperative day (pulmonary embolus) |
| 197 | Armanino [2] | 1946 | M | 17 | + | + | + | Cirrhosis of liver or possibly primary carcinoma of liver | No surgical treatment | Died on 5th day after admission; postmortem: metastatic carcinoma, congenital cystic dilatation |
| 198 | McLaughlin [43] | 1946 | M | 22 | — | — | + | Suspicion of congenital cystic dilatation of common bile duct, first among possible diagnoses | Removal of 9/10 of cystic dilatation and anastomosis of the remnant of cystic dilatation to duodenum | Recovery |
| 199 | Kelsey [36] | 1947 | F | 4 | + | + | — | Choledochus cyst (?) | Choledochocystoduodenostomy | Recovery |
| 200 | Fleming [21] | 1947 | F | 29 | + | — | — | (Cholecystitis ?); pyloric obstruction | Choledochocystoduodenostomy | Recovery |
| 201 | Keating & Murphy [34] | 1947 | F | 2½ | — | + | + | Semicystic tumor of right upper quadrant of abdomen | Choledochocystoduodenostomy | Died on 31st postoperative day |
| 202 | Stajano [68] | 1946 | F | 25 | + | — | + | Hydatid cyst of liver | 1. Drainage of congenital cystic dilatation; 2. 14 days later, partial excision of congenital cystic dilatation; choledochogastrostomy | Recovery |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 203 | Ripstein & Miller [58] | 1948 | F | 4 mo. | — | + | ? | Congenital atresia or choledochus cyst | Excision of congenital cystic dilatation; inoperable case, because of coexistent atresia of hepatic and bile ducts<br>Operative findings: congenital cystic dilatation associated with congenital atresia of bile ducts | Died |
| 204 | Urwitz [75] | 1948 | M | 9 | — | + | + | Congenital choledochus cyst | Cholecystostomy | Died on 8th postoperative day |
| 205 | Keeley [35] | 1948 | M | 30 | + | + | — | Cholecystitis | Anastomosis, Roux-en-Y type, between congenital cystic dilatation and jejunum | Recovery |
| 206 | Davis [17] | 1948 | M | 5 | + | + | + | A. On first admission, acute hepatitis; B. on 2d admission, choledochus cyst | Aspiration and biopsy of congenital cystic dilatation; biopsy of liver; choledochocystoduodenostomy | Recovery |
| 207 * | Kjellberg [37] | 1948 | M | 7 | — | — | + | Cyst of the common bile duct | Drainage only | Died; postoperative peritonitis |
| 208 | Rogers & Priestley [59] | 1949 | F | 62 | + | + | + | Not mentioned | Drainage of the congenital cystic dilatation and Roux-en-Y type choledochocystojejunostomy | Recovery |
| 209 | Archambault, Archambault, & Lasker [1] | 1950 | F | 17 | + | + | + | Abnormally located appendicitis or cholecystitis | Choledochocystojejunostomy; anastomosis Roux-en-Y type | Recovery |
| 210 | Lamprecht [40] | 1950 | M | 16 | + | + | — | 1943, appendicitis;<br>1946, intestinal obstruction, cholangitis;<br>1947, choledochus cyst | Appendectomy<br>Drainage of congenital cystic dilatation<br>Choledochocystoduodenostomy | Recovery<br>Recovery<br>Recovery |
| 211 | Huang & Tsui [30] | 1950 | F | 3 mo. | — | + | + | 1. Infectious hepatitis<br>2. Date of admission uncertain | Aspiration<br>Choledochocystoduodenostomy | Died on first postoperative evening |
| 212 | Simon [65] | 1950 | F | 3½ mo. | + | + | + | Hepatic cirrhosis (?) | Aspiration; choledochocystogastrostomy | Died on 4th postoperative day |
| 213 | Vogogna & Larre [77] | 1950 | M | 21 | + | + | + | Choledochus syndrome m. insufficiency of the liver, obstructive jaundice | 1. Marsupialization and drainage of congenital cystic dilatation;<br>2. after 3 mo., choledochocystoduodenostomy | Recovery |
| 214 | Case Records of Mass. General Hospital [12] | 1951 | F | 15 | — | + | + | Clinical diagnosis: enterogenous cyst (duplication of duodenum); Dr. Welch's diagnosis: congenital dilatation of common bile duct | Choledochocystojejunostomy (Roux-en-Y); drainage of congenital cystic dilatation | Recovery |
| 215 | Macpherson [44] | 1951 | F | 17 | + | — | — | Postpartum mortality following premature delivery; clinical diagnosis: prerenal uremia from persistent vomiting; postmortem: large cystic dilatation of common bile duct (about 20 cm.×11 cm. containing 3 to 500 cc. of normal-looking bile) | | |
| 216 | Kong [38] | 1951 | M | 54 | + | — | ? | Perforation of appendix with peritonitis | Aspiration; marsupialization and drainage | Recovery |
| 217 | Helfferich [28] | 1951 | F | 24 | + | — | — | Ulcer of duodenum | No operation | Died; postmortem: congenital cystic dilatation communicating with pancreatic duct and rupture of the aorta |
| 218 | de Quadros [55] | 1952 | F | 38 | + | + | — | ? | Cholecystectomy, choledochoduodenostomy, drainage | Recovery |
| 219 | Gale & Peterson [23] | 1950 | F | 15 | + | + | — | Meckel's diverticulum, or chronic appendicitis, or peptic ulcer | Aspiration of congenital cystic dilatation; excision of anterior wall of cystic dilatation and reconstruction of common bile ducts; cholecystectomy | Recovery |

TABLE 8.—*Congenital Cystic Dilatation of the Common Bile Duct: Fifty-Seven Cases Not Analyzed in Previous Reviews*—Continued

| No. | Author | Year of Publication | Sex | Age, Yr. | Symptoms | | | Preoperative Diagnosis | Treatment | Results and Follow-Up |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Pain | Jaundice | Tumor | | | |
| 220 | Hertzler & Maquire [29] (1st case) | 1951 | F | 5 | + | — | + | No diagnosis ? | Marsupialization and secondary choledochoecystoduodenostomy | Recovery |
| 221 | (2d case) [29] | 1951 | M | 1 | + | + | + | Diffuse infiltration of right kidney or possibly pressure from adjacent soft-tissue mass | Excision of congenital cystic dilatation and cholecystectomy; choledochoduodenostomy | Recovery |
| 222 | Strohl & Sarver [69] | 1951 | F | 24 | + | + | — | Congenital dilatation of common bile duct (?) | 1. Cholecystectomy, drainage of congenital cystic dilatation; 2. 3 mo. later, choledochocysto-jejunostomy Roux-en-Y type | Recovery |
| 223 | Gilliland, Holloway, & Lange [25] | 1950 | F | 4 mo. | — | + | + | Congenital choledochus cyst (first among preoperative diagnoses) | Side-to-side anastomosis of congenital cystic dilatation and duodenum | Recovery |
| 224 | Sullivan & Dmytryk [70] | 1951 | M | 12 | + | + | — | Pathologic condition in duodenum beyond bulb | Excision of congenital cystic dilatation and cholecystectomy; choledochojejunostomy (4 in. lower) | Recovery |
| 225 | Newson [50] | 1952 | F | 12 | — | + | + | Hydatid cyst (?) | 1. Anastomosis of congenital cystic dilatation with pyloric portion of stomach; 2. posterior gastroenteroanastomosis (because of pyloric obstruction) | Recovery |
| 226 | Garces, Neira, & Guzman [24] (1st case) | 1952 | M | 8 mo. | + | + | ? | Obstructive jaundice; cirrhosis of liver | No operation | Died on 9th postoperative day of bilateral bronchopneumonia and biliary cirrhosis of liver; postmortem: congenital cystic dilatation (250 cc.); cirrhosis of liver |
| 227 | (2d case) [24] | 1952 | M | 3 mo. | — | + | ? | Peritonitis, hepatitis; radiologically, cystic abdominal tumor | No operation | Died on 8th day after admission; postmortem: rupture of congenital cystic dilatation and biliary peritonitis |
| 228 | (3d case) [24] | 1952 | F | 1 yr. 4 mo. | — | + | + | 1. Mesenteric cyst; 2. cystic abdominal tumor localized in pancreatoduodenal region | Choledochoecystoduodenostomy | Recovery |
| 229 | Dickinson & Spencer [18] | 1952 | F | 2 mo. | ? | + | ? | Biliary atresia or choledochal cyst | Choledochoecystoduodenostomy | Recovery |
| 230 | Authors' cases‡ (1st case) | .... | F | 27 | + | + | — | Cholecystitis | 1. Choledochoecystoduodenostomy; 2. choledochoecystojejunostomy Roux-en-Y type | Recovery |
| 231 | (2d case)‡ | .... | F | 34 | + | — | — | Chronic recurrent pancreatitis | 1. Partial resection of congenital cystic dilatation and reconstruction of common bile duct; transduodenal sphincterotomy; cholecystectomy; 2. choledochoduodenostomy for choledocholithiasis and stricture of common bile duct | Recovery |
| 232 | (3d case)‡ | .... | M | 58 | + | + | — | Choledocholithiasis; cholecystitis | Choledochoecystoduodenostomy | Recovery |

* This case is not included; it is referred to only for the follow-up.
† In cases 181, 182, and 183 the disease was discovered only at postmortem examination.
‡ Cases of Dr. W. Davis, Dr. George Crile Jr., and Dr. S. O. Hoerr, respectively.

but they never connote "cystic." Thus, the compound word "megalocholedochus" does not characterize the condition of the bile duct in which the wall is enlarged equally all along its length, and it does not denote the cystic dilatation of the duct and does not connote the origin. The term "diverticulum of the common bile duct" is incorrect because the lesion involves the entire circumference and not just a portion of the wall. The term "congenital dilatation of the hepatacholedochus" does not indicate the condition as arising from the common bile duct and only in the part which is 2 cm. above the duodenum. The term "congenital cyst of the common bile duct" is not accurate because this condition is not a cyst but a cystic dilatation, congenital in origin. The most suitable term that meets all the requirements of a clear-cut characterization is the term "congenital cystic dilatation of the common bile duct."

## SUMMARY

Data from 232 cases of congenital cystic dilatation of the common bile duct are tabulated. Three new cases are included. The congenital origin is favored, and the most plausible explanation is that of unequal proliferation of the epithelial cells at a fetal stage of pathologic epithelial occlusion of the duct. Knowledge of the pathology is amplified by the data supplied by the 57 analyzed cases. The diagnosis may be suspected in patients exhibiting the triad of "pain, jaundice, tumor" and is established by operation. Employment of operative cholangiography should assist in accurate surgical diagnosis.

The mortality rate of 84% reported in the first cases [41] has now been reduced to 31.5% in the most recent 57 cases. Better diagnosis, better preoperative and postoperative care, and the performance of choledochocystoenterostomy, either between cystic dilatation and duodenum or a Roux-en-Y type to jejunum, should reduce the mortality rate even further. Choledochocystojejunostomy is considered as the method of choice.

The terminology of the condition is discussed, and reasons are given for favoring the term "congenital cystic dilatation of the common bile duct."

Cases of Drs. W. Davey, of Whittington Hospital (London), George Crile Jr., and Stanley O. Hoerr, of Cleveland Clinic Hospital, were included in this study.

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* This case is not included; it is referred to only for the follow-up.
† In cases 181, 182, and 185 the disease was discovered only at postmortem examination.
‡ Cases of Dr. W. Davis, Dr. George Crile Jr., and Dr. S. O. Hoerr, respectively.

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# Five-Year Survival After Hepatic Lobectomy for Metastatic Hypernephroma

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Hypernephroma reputedly produces more solitary distant metastases than any other malignant tumor. The primary site is frequently silent, and the diagnosis is frequently made from a study of the distant implant. Apparent isolated metastases have been described in the lung,[1] brain,[2] bone,[3] pancreas,[4] thyroid,[5] testis,[6] and vagina.[7]

An occasional attempt has been made at resection of the metastasis. In some instances the operator thought he was treating a primary tumor; in others the primary tumor seemed to be under control and an attack on a solitary metastasis appeared justified. Sometimes this appears to have been successful.

Herbst [8] resected an isolated metastasis in the region of the duodenal papilla, and the patient was well six months later. Jenssen's [4] patient was well nine months after resection of an implant in the head of the pancreas. Riches, Griffiths, and Thackray [9] report removal of apparent solitary metastases in seven cases followed four months to one year. Edelman's [2] patient had resection of a tumor in the cerebellum followed by nephrectomy; the patient died three years later of carcinomatosis.* Wishner † reported a case of interscapulothoracic amputation for metastatic hypernephroma; the patient died three years later of pulmonary tuberculosis. He probably also had pulmonary metastases. Ewert's [11] case of pulmonary lobectomy was free of tumor for at least three years. Dempsey, Crile, and Engel ‡ reported a thyroidectomy followed by nephrectomy. Their patient was well five years after operation. Albrecht had two cases of long survival after resection of a solitary metastasis. One patient § was well five years after amputation of an implant in the femur; the patient refused nephrectomy. The other patient [3] was well nine years after resection of a scapular metastasis. Bandler and Roen [6] report a case of orchidectomy in which a nephrectomy was done two years later, and the microscopic picture of the two lesions was the same. This patient died of unknown cause seven years after orchidectomy. Barney [1] reported a case in which the patient was well 11 years after pulmonary lobectomy for a metastasis. Bumpus,[13] Beer,[14] and Mann ‖ have all reported cases of spontaneous disappearance of pulmonary metastases from hypernephroma, but in each instance the diagnosis was made from x-rays

Submitted for publication April 6, 1955.

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* Edelman, L.: Personal communication to the authors.

† References 10 and Wishner, J. C.: Personal communication to the authors.

‡ Reference 5 and Crile, G., Jr.: Personal communication to the authors.

§ Albrecht, P., cited by Tavernier.[12]

‖ Reference 15 and Mann, L. T., cited by Ackerman and del Regato.[16]

of the chest alone; histological proof is lacking.

REPORT OF A CASE

A woman aged 62 was admitted to the Presbyterian Hospital on March 6, 1950, with a three-month history of burning in the stomach and progressive abdominal swelling; she had had fever for four days. Her history disclosed that in 1945 she had a left nephrectomy for hypernephroma.

Significant physical findings included a blood pressure of 160/80. In the upper abdomen there was an obvious mass the size of a large grapefruit, with an ill-defined border that extended down to the umbilicus in the midline. Fluoroscopy showed

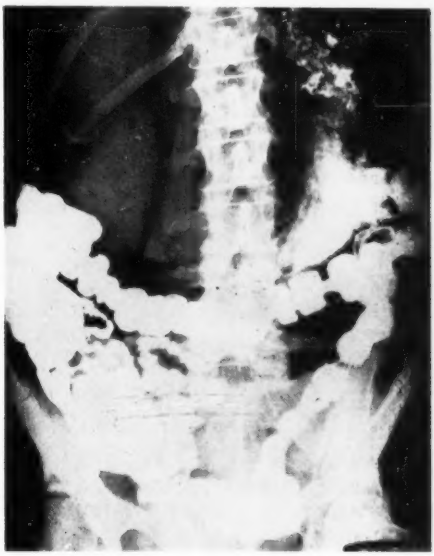


Fig. 1.—Fluoroscopy demonstrates the colon to be displaced down and the stomach to be displaced down and to the left by an epigastric mass.

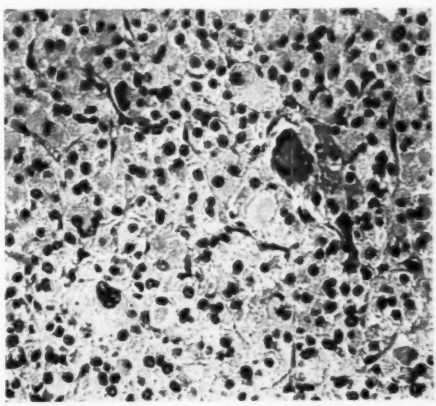
the transverse colon to be displaced down and the stomach to be displaced down and to the left (Fig. 1). Urinalysis and blood cell count were normal. Results of blood chemistry studies were as follows: NPN 46 mg., alkaline phosphatase 5.7 units, icteric index 10, albumin-globulin ratio 4.3: 3.4 gm/100 cc., cephalin flocculation negative at 24 and 48 hours, and prothrombin time 75%. Blood sugar after fasting was 128 mg/100 cc., and a glucose-tolerance test showed a diabetic curve.

The patient was operated upon on March 10, 1950. The only evidence of pathology in the abdomen was a large bosselated tumor occupying almost the entire left lobe of the liver. A small area

Fig. 2.—The gross specimen of tumor in the left lobe of the liver weighed 1500 gm.

of normal liver tissue extended between the tumor and the falciform ligament. Two parallel layers of large overlapping absorbable surgical (gut) mattress sutures were placed immediately adjacent to the falciform ligament. The liver was then cut across in the plane between the suture layers. There were a few moments of brisk bleeding, but hemostasis was effected with clamps and hot packs. During manipulation at this time, the tumor was pushed against the costal margin and the capsule was broken. Presumably, free tumor cells were deposited in the peritoneal cavity. After all bleeding

Fig. 3.—Microscopic appearance of metastatic hypernephroma in the liver.



points were ligated, the lower edge of the right lobe was sutured to the upper. The peritoneum was irrigated with saline solution, and the wound was closed. Blood replacement at operation was 5 pt. Convalescence was uneventful, and the patient went home on the ninth postoperative day. The pathology report ¶ describes a tumor which weighed 1500 gm. (Fig. 2). Protruding from the surface were several lobulations, each measuring 6 cm. in diameter. Microscopic sections revealed an extremely malignant, rapidly growing, and invasive metastasis from a hypernephroma (Fig. 3).

For three years after operation, the patient felt well. Two years ago she had an episode which suggested a mild attack of coronary thrombosis. Since that time she has become fatigued more easily, but otherwise has been well. Now, at age 67, she still does all of her own housework.

Physical examination at the present time is vitually unchanged from the immediate postoperative period. There is no evidence of recurrence of her tumor. Results of blood chemistry studies are about the same as before operation: NPN 42 mg., albumin-globulin ratio 4.7 : 2.7 gm., and blood sugar after fasting 130 mg/100 cc. and cephalin flocculation negative at 24 and 48 hours. Her chest x-ray is normal.#

It is well known that in dealing with hypernephroma a five-year survival is not tantamount to cure. Some cases of long survival without any treatment have been reported. Denton and McClintock [17] report a case in which the first metastasis appeared in the thyroid eight years after nephrectomy. Caylors' case [18] of metastasis to the thyroid had a 13-year interval between nephrectomy and appearance of the secondary tumor.

One of us (F. H. S.) had a patient whose symptoms of hematuria, dysuria, and fatigue began in 1923. In 1928 a nephrectomy was done for hypernephroma. In 1934 a hysterectomy was done, and no other abnormalities were noted. The patient remained well for 19 years. In December, 1946, symptoms of recurrence appeared and a liver biopsy showed metastatic hypernephroma. She died in March, 1949, twenty-six years after the onset of symptoms and 21 years after nephrectomy.

Despite the possibility that the patient being studied may still succumb to metastatic hypernephroma, it is self-evident that the procedure has been well worth while. Her general health is still better than it was preoperatively. In addition, the microscopic appearance and the history of rapid enlargement would suggest that her course would have been different had her tumor not been resected.

¶ George M. Haas, Chairman, Department of Pathology, Presbyterian Hospital.

# This patient was last seen Jan. 12, 1956. At that time examination revealed no evidence of metastases. X-ray films of her lumbar spine showed osteoarthritic changes, but no evidence of metastasis. X-ray films of her pelvis and her right femur were normal.


## SUMMARY

A review is made of survivals after resection of solitary metastasis from hypernephroma.

A case of five-year survival after left hepatic lobectomy for metastatic hypernephroma is presented.

A case of hypernephroma is reported in which the metastases first became evident 21 years after nephrectomy.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# Necrosis Following Administration of Levarterenol During Shock

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We have recently seen seven instances of tissue necrosis when levarterenol (Levophed) was used to raise the blood pressure during a period of acute hypotension. Similar cases have been previously reported, and we are aware of many unreported instances. In view of the widespread use of vasopressor agents to combat shock, one may expect an increased number of such complications in the future, unless more caution is observed.

The mechanism by which levarterenol raises blood pressure is probably that of generalized vasoconstriction producing increased peripheral resistance. One might possibly explain the occurrence of local tissue necrosis following the intravenous administration of levarterenol on the basis of intense vasospasm. This spasm is manifested not only in the smaller arteries and veins but also in the vasa vasorum. It is possible that local tissue irritation in association with extravenous placement of the solution may be a factor. These two mechanisms might act to impair local tissue oxygenation, resulting in gangrene.

The following case reports are illustrative of the problem.

## REPORT OF CASES

CASE 1.—A 52-year-old white woman was admitted to the medical service of the St. Louis City Hospital on Dec. 2, 1951, in a state of hypotension associated with posterior myocardial infarction and complete heart block. The systolic blood pressure on entry was 60 mm. Hg.; the diastolic, 50 mm. Hg. Fifty minutes after admission a polyethylene catheter was threaded into the surgically exposed long saphenous vein, at the right ankle. Through this catheter a solution containing 2.0 cc. of levarterenol in 1000 cc. of 5% dextrose in water was given slowly. After 90 minutes of this therapy the blood pressure had risen to 120/80 mm. Hg., where it was maintained throughout the day by infusion of the levarterenol solution at a rate of 8 to 30 drops a minute. This therapy was discontinued and the catheter removed after 14 hours because of "infiltration" in the area around the phlebotomy site. The overlying skin was edematous, cold, and white. The following morning a well-demarcated reddish-purple area with vesicle formation was present, accompanied by extreme pain. An area measuring 7 by 20 cm. near the medial malleolus of the leg was subsequently débrided and grafted. The graft healed well, and she was discharged on Feb. 8, 1952.

CASE 2.—A 29-year-old white woman was admitted to the medical service of the St. Louis City Hospital acutely ill with septicemia due to endometritis. The blood pressure on entry was 70/30 mm. Hg. Hypotension persisted despite supportive therapy. A blunt cannula was inserted into the surgically exposed long saphenous vein at the ankle. Through this cannula she was given slowly a solution of 5% dextrose in water containing 4 cc. of levarterenol a liter. By this means the systolic blood pressure was maintained at about 100 mm. of Hg. for the next 26 hours. She then developed pallor of the skin and multiple bullae over the lower anteromedial aspect of the left leg. There was no evidence of subcutaneous extravasation. The "cut-down" was discontinued, and hot packs were applied. A painful 12×12 cm. area ultimately sloughed and was skin-grafted after 6 weeks.

Because of hypotension after discontinuance of the infusion in the leg, the same solution was given through an intravenous needle in the right antecubital fossa. Infiltration occurred after several hours and was followed by slough 4 cm. in diameter. Bismuth tribromphenate (Xeroform) gauze dressings were used, with complete healing in 7 weeks.

CASE 3.—A 68-year-old white woman was admitted to the gynecology service of St. Mary's Hospital on Nov. 23, 1953, with a diagnosis of

Submitted for publication April 12, 1955.

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procidentia and perineal relaxation. Vaginal hysterectomy and perineorrhaphy were performed under thiopental (Pentothal)-nitrous oxide-oxygen anesthesia. Postoperatively, in an effort to combat shock, an infusion of 4 cc. of levarterenol in 1000 cc. of 5% dextrose in water was started in a vein on the dorsum of the left hand. Subcutaneous extravasation occurred shortly thereafter, and the infusion was discontinued. A solution containing 50 cc. of 1% procaine and 500 viscosity units of hyaluronidase was injected into the area of infiltration, and no sequelae were noted.

Several days later the patient again became hypotensive. By way of a cannula placed in the exposed long saphenous vein at the right ankle, she was given in eight hours a solution containing 4 cc. of levarterenol in 1000 cc. of 5% dextrose in water. This was followed by fluids containing no levarterenol. Twelve hours later a red, warm area was noted above the ankle. A solution of 50 cc. of 1% procaine and 500 viscosity units of hyaluronidase was injected into the tissue of this area. Several days later a large, irregular, well-demarcated zone of gangrene became apparent about the lower anteromedial aspect of the right leg. This was later skin-grafted with satisfactory healing.

Case 4.—A 29-year-old white woman was admitted to Firmin Desloge Hospital with an external small bowel fistula. At operation the fistula was excluded and small intestine continuity restored. The patient returned to the ward in a hypotensive state and was given intramuscular doses of methoxamine hydrochloride (Vasoxyl) every four hours. Her systolic blood pressure ranged from 60 to 80 mm. Hg. for 12 hours, after which a slow drip of 2 cc. of levarterenol added to 1000 cc. of 5% dextrose in water was infused through a cannula placed into the left long saphenous vein at the ankle. This was followed by a solution containing 4.0 cc. of levarterenol diluted in 1000 cc. of 10% dextrose in water. After 700 cc. of this solution had been given during 8 hours, subcutaneous extravasation was noted and the cannula was removed. The overlying skin was blanched, tender, and cold. Four hours later hypotension again ensued, and the remaining levarterenol solution was given without incident through an intravenous needle placed percutaneously in the antecubital fossa.

Within 12 hours after discontinuation of levarterenol a well-demarcated painful area of purplish discoloration with superficial blebs became apparent in the area of infiltration about the medial aspect of the left ankle and dorsum of the foot. An ulcerated, sloughing area measuring 10×20 cm. gradually developed. This was débrided and skin-grafted with primary healing. No evidence of tissue necrosis was noted about the antecubital fossa (Figure).

Case 5.—A 54-year-old white man was admitted to the St. Louis City Hospital, where an elective mastoidectomy was performed with general anesthesia.

In the immediate postoperative period, with a systolic blood pressure of 60 mm. Hg., a solution containing 12 cc. of levarterenol diluted in 1000 cc. of 5% dextrose in water was administered through a needle placed into an antecubital vein. Infiltration occurred at the site of venepuncture; a polyethylene catheter was placed into the right long saphenous vein at the ankle, and the levarterenol solution was given here. This had to be discontinued several hours later because of local pain, blanching, tenderness, and edema. A "cutdown" was performed in the left saphenous vein, at the ankle, and the levarterenol solution was continued for 15 hours. There was no extravascular leakage demonstrable at the left ankle.

Figure—Full thickness skin slough following subcutaneous extravasation of levarterenol.

An irregular 8×15 cm. area of tissue slough developed over the medial aspect of the right lower leg, which required skin grafting. A 5×8 cm. area of ischemia above the left ankle was followed by crusting and, later, spontaneous healing. There was no evidence of cutaneous ischemia in the area of extravasation in the antecubital fossa.

Case 6.—A 27-year-old white man was admitted to the St. Louis City Hospital in coma after a head injury. During the next few days his course

was a downhill one. On the fourth day he went into shock with systolic blood pressure of 60 mm. Hg. A "cut-down" was performed and a polyethylene catheter was threaded into the right long saphenous vein at the ankle. A liter of 5% dextrose in water with 24 cc. of levarterenol added was started as a slow drip. Nine hours later subcutaneous extravasation was noted and the catheter removed. The overlying skin was tense, edematous, cold, and pale. Ice bags were applied to the area. The following day an irregular 20×12 cm. area of purplish discoloration was noted above the right medial malleolus. One week later frank laceration of the skin and subcutaneous tissue developed.

On discontinuing the "cut-down" in the right leg, another "cut-down" was placed in the left ankle and a polyethylene catheter threaded to just below the knee. During the next four days varying dilutions of levarterenol were infused. The infusion was finally discontinued because of a 10×7 cm. area of purplish discoloration of the skin below the medial aspect of the left knee. There was no evidence of infiltration. The patient died shortly thereafter.

CASE 7.—An 87-year-old white woman developed severe hypotension immediately after laparotomy for relief of intestinal obstruction due to adhesions. A polyethylene catheter was placed into the surgically exposed long saphenous vein at the right ankle, and parenteral fluids were started. A blood pressure was maintained between 80 and 90 mm. Hg. systolic. The following morning 4 cc. of levarterenol was added to 1000 cc. of 5% dextrose in water and infusion continued. For the next 30 hours the patient received a continuous drip of a solution containing between 4 and 8 cc. of levarterenol per liter of 5% dextrose in water. The solution was discontinued because of subcutaneous extravasation. The overlying skin of a 10×25 cm. area along the anteromedial aspect of the lower leg was cold and white. Fifty cubic centimeters of 1% procaine solution was promptly injected into the area. Later that day a well-demarcated area of purplish discoloration of the skin with superficial blebs became apparent. Frank necrosis and gangrene developed during the next few days and persisted until the patient's death shortly thereafter.

Histological sections through the gangrenous area and about the periphery revealed extensive thrombosis of both arteries and veins involving vessels of all calibers. The lining of the blood vessels displayed marked edema of the media.

## COMMENT

The cases presented here and those previously reported have certain features in common. The majority of ulcerations have occurred after intravenous infusion about the ankles. Subcutaneous extravasation is not requisite for ulceration. Sloughs have occurred with both metal cannulas and polyethylene catheters. The ulceration in the upper extremity reported here is the second such instance of tissue necrosis recorded in this region after extravasation during infusion of levarterenol.

These observations lead us to believe that some factor other than local tissue irritation is responsible for ulceration. On the basis of experimental observations it is our belief that peripheral ischemia induced by shock is aggravated by levarterenol under the conditions noted. This intereferes with cellular nutrition and may result in tissue necrosis and gangrene.

Assuming that this mechanism exists, what therapeutic program should be outlined? First, one should exert caution in the use of vasopressors in acute hypotensive states. Care should be taken to insure against extravenous leakage of infusions containing these agents. As soon as evidence of cutaneous ischemia or subcutaneous extravasation is detected, the infusion should be immediately discontinued.

We have tried several agents in local therapy of this complication. It is our clinical impression that the most effective local treatment includes infiltration with a mixture of procaine and hyaluronidase. The former agent may counteract local vasospasm and the latter should facilitate resorption of extravasated solution. This is not a uniformly successful treatment, however.

Investigation of the pathogenesis and therapy of this condition is continuing. In view of the complications mentioned, it appears that the widespread, uncritical use of vasopressor agents to counteract hypotension without treating its basic cause is unwise.

## SUMMARY

We have reported seven instances of tissue necrosis following the intravenous administration of levarterenol during hypotensive states.

Analysis of these cases and others, combined with limited experimental investigation,

suggests that the cutaneous ischemia accompanying shock may be aggravated by the intravenous use of certain vasopressor agents. The resulting interference with tissue metabolism may lead to tissue necrosis and gangrene.

Investigations are being continued to elucidate the pathogenesis and therapy of this complication. A few tentative therapeutic suggestions have been presented.

The need for caution in symptomatic treatment of acute hypotension is stressed.

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# Metabolic Derangements Imperiling the Perforated Ulcer Patient

## IV. Derangements of Nitrogen Metabolism and the Nitrogen Deficit

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### INTRODUCTION

It is generally accepted that trauma disturbs nitrogen metabolism.* Reported first as a sequel of a fracture, the disturbance follows a burn and a variety of operations. The obvious manifestation of this disturbance is an increased excretion of nitrogen in the urine. After minor trauma, this excretion persists for only a few days; after severe trauma, for four weeks or longer. Unless this abnormal excretion can be counterbalanced by an increased intake, the patient suffers a nitrogen deficit. To a previously healthy person a transient deficit may be of little moment, but to those who have been ill and debilitated the deficit may lead to a crippling nitrogen depletion.

Questions regarding three aspects of the disturbance in nitrogen metabolism prompted this study in the patient with the perforated peptic ulcer: the extent of the disturbance, the effect of any deficit upon the clinical course, and the management of such a deficit. Howard has suggested that the outpouring of nitrogen in the urine following trauma is the reaction of the previously healthy person.[5] He has found no increase above the expected normal in a cachectic patient who fractured a femur, and a similar observation has been made at this hospital in a severely burned woman. She had been chronically ill with tuberculosis when burned. This observed difference between the healthy and the ill raised the question of the category to which the patient with the perforated ulcer belongs. The ulcer patient has been off his feed and under stress, often for some time before perforation. Depletion is the rule, and, to bear this out, the patients of this series proved to be underweight. The 24 men averaged only 127 lb. (58 kg.) on admission.

The importance of maintenance of nitrogen balance to wound healing, immune body production, and prompt return to well-being has been much discussed but is still incompletely understood. The achievement of nitrogen balance in patients with perforated ulcers is delayed because of the very position of the lesion in the gastrointestinal tract. Parenteral nitrogen is, therefore, essential if a deficit is to be wiped out. What form of nitrogen is to be used? Concentrated albumin and plasma are expensive, and their nitrogen may not be immediately available to the intermediary metabolism. The literature bristles with conflicting reports concerning the effectiveness of intravenously administered amino-acid hydrolysates. Some investigators report quantitative excretion of amino-acid nitrogen, whereas others report its retention.† A possible explanation of these conflicting reports was put forth by Peters, who suggested that a depleted man may be capable of utilizing protein products

Submitted for publication April 20, 1955.

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This work was supported by a contract between the Office of Naval Research and Harvard University.

* References 1 to 10.

† References 2, 4, 8, and 11 through 19.

which a healthier person would excrete.[20] This concept, particularly in regard to the patient with a perforated ulcer, seemed to merit further investigation.

## METHODS

The main features of the investigative routine are described in the first paper of this series.[21] Intake data were derived from weights or careful volumetric measurements, as described in the second paper.[22] Urine, stools, gastric contents, and peritoneal fluid were analyzed for nitrogen. Total nitrogen determinations were done by the Kjeldahl method [23] and nonprotein nitrogen by a modified Folin-Wu technique.‡

## PLAN OF STUDY

In this study of nitrogen deficit and utilization the patients were divided into two groups, those receiving a low intake of nitrogen during the first week after perforation and those with a high intake. The patients on the low intakes received as their only nitrogen that in the whole blood needed for shock or anemia. In the high intake group nitrogen was added by the intravenous administration of amino-acid hydrolysates. No plasma was given to either group.

The resumption of oral feedings was variable both as to time and as to nitrogen content.

‡ References 24 and 25.

## OBSERVATIONS

### A. Nitrogen Balance

The nitrogen balance was studied in 17 patients. There were 10 in the low-intake group and 7 in the high. The low-intake group received an average of 2.1 gm. a day for the first week. In the high-intake group in the same period, five patients received between 5 and 10 gm. a day and two received more than 10 gm. daily. The single case without operation is included in the low-intake group.

The study reveals a trend of initial nitrogen loss and subsequent retention. It was found possible to reduce the nitrogen deficit by the intravenous administration of amino-acid hydrolysates.

1. *The Nitrogen Deficit.*—(a) Nitrogen Wasting in the Absence of Nitrogen Therapy: In the nine patients who were operated on and received no amino-acid hydrolysates, nitrogen output exceeded intake for 8 to 10 days after perforation (Chart 2). During the first week the average daily deficit in the seven patients with complete balance studies was 8.0 gm. (Table 2). The cumulative deficit was large, amounting in the average case to 56 gm. on the 10th postoperative day.§

§ A loss equivalent to 350 gm. of protein, or 1.75 kg. of body tissue.

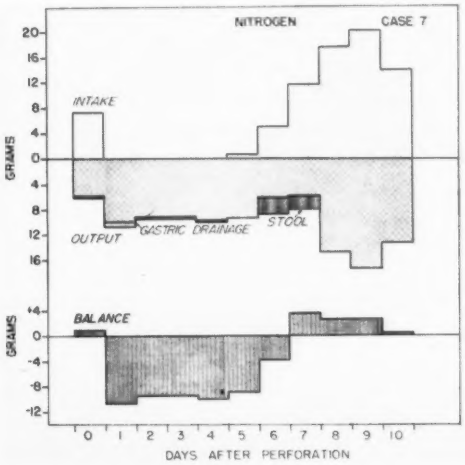


Chart 1 (Case 7).—Initial nitrogen depletion and subsequent retention in a patient from whom intravenous nitrogen therapy was withheld. The patient was given a single transfusion on the day of perforation, to combat shock, but otherwise received no nitrogen by vein. A nitrogen deficit developed postoperatively which persisted until oral feeding raised the patient's nitrogen intake above 10 gm. a day. The changes are typical of the low-intake cases.

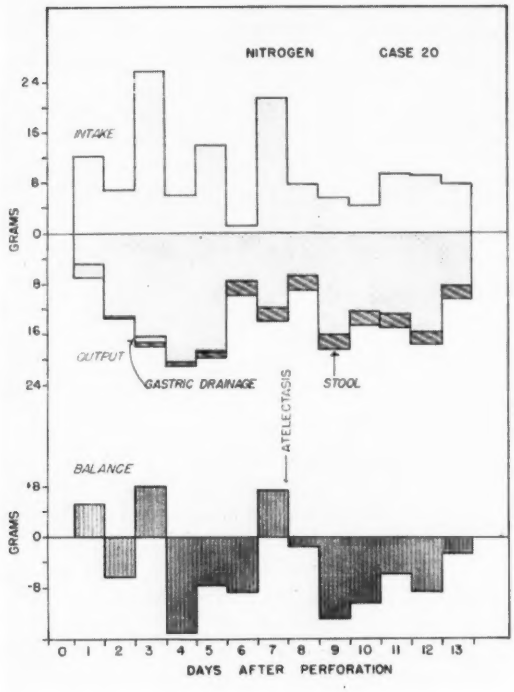


Chart 2 (Case 20).—Diminution of the postoperative nitrogen deficit and reversal of negative balance by intravenous amino-acid therapy. Administration of moderate to large infusions of amino-acid hydolysates on the first, third, fifth, and seventh postoperative days increased the nitrogen output but simultaneously decreased or reversed the negative nitrogen balance. No attempt was made to supplement oral nitrogen intake during the second week. The patient, an asthmatic, developed atelectasis of the lower lobe of the left lung, with elevation of temperature to 103 F on the seventh day after perforation. His oral nitrogen intake diminished and urinary output increased. He remained in negative nitrogen balance from the 8th through the 13th postoperative day.

TABLE 1.—*Urinary Nitrogen Excretion in Healthy Subjects on Various Nitrogen Intakes (Data from the Literature)*

| Source | Number of Subjects | Condition of Subjects | Comments Concerning Intake | Av. Nitrogen Intake/ Day, Gm. | Av. Urinary Nitrogen Output/ Day, Gm. |
|---|---|---|---|---|---|
| 26 | 12 | Healthy males | Unrestricted | 15.5 | 12.9 |
| 27 | 10 | Healthy males | Controlled | 16.5 | 15.6 |
| 26 | 12 | Healthy males | First week of restriction | 11.5 | 12.1 |
| 26 | 12 | Healthy males | Early in restriction period | 8.2 | 10.4 |
| 26 | 12 | Undernourished but otherwise healthy | After prolonged restriction | 11.0 | 10.9 |
| 27 | 10 | Undernourished but otherwise healthy | After 11 weeks of semistarvation | 7.6 | 8.1 |
| 27 | 10 | Undernourished but otherwise healthy | After 23 weeks of semistarvation | 7.8 | 7.5 |
| 28 | 1 | Fasting | First weeks of absolute fast | 0.0 | 9.9 |
| 28 | 1 | Fasting | Last week of 31-day fast | 0.0 | 7.7 |

The single patient who did not come to operation remained in negative nitrogen balance throughout the 10 days he was under study, losing 133 gm. of nitrogen, 55 gm. more than were lost by any of the surgically treated cases.

A surprising feature of the postoperative period was the low renal output of nitrogen

TABLE 2.—*Urinary Nitrogen Excretion and Nitrogen Deficits in Perforated Ulcer Patients on Various Nitrogen Intakes (Data from this series)*

| Number of Patients | Amino-Acid Hydrolysates | Av. Nitrogen Intake/ Day, Gm. | Av. Urinary Nitrogen/ Day, Gm. | Total Nitrogen Output/ Day, Gm. | Nitrogen Deficit/ Day, Gm. |
|---|---|---|---|---|---|
| 7 | None | 2.9 | 9.6 | 10.9 | 8.0 |
| 1* | None | 1.9 | 14.8 | 15.7 | 13.8 |
| 5 | Given irregularly or in small amounts | 5.6 | 11.8 | 12.5 | 6.9 |
| 2 | Given in large amounts | 20.1 | 20.9 | 22.0 | 1.9 |

* Case treated without operation.

by these patients (Tables 1 and 2). The urinary nitrogen outputs average 9.6 gm. a day (0.4 gm. an hour) throughout the first week, except on the day of perforation and operation. Elevations in rate above this during the immediate postoperative period were slight and fleeting, averaging 0.7 gm. an hour for 14 hours. Of three patients on nitrogen-free intakes during the day of perforation, one showed no hourly output above 0.4 gm., one averaged 0.5 gm. for the day, and the third exhibited an increase to 0.9 gm. for 12 hours.

(b) The Expensive Economy of Intravenous Amino-Acid Therapy: Administration of amino-acid hydrolysates by vein reduced the nitrogen deficit, even though it increased urinary excretion. In five cases receiving amino acids irregularly or in small quantities, the therapy reduced the daily deficit from 8.0 to 6.9 gm. (Table 2). In two cases moderate to large infusions given regularly reduced the deficit to 1.9 gm. This diminution in nitrogen depletion occurred despite an outpouring of urinary nitrogen during the amino-acid therapy. The daily output of the two patients receiving the largest amounts a day (average, 20.1 gm.) averaged 20.9 gm. In contrast, the group receiving no amino-acid nitrogen excreted an average of only 9.6 gm. (Table 2).

2. *Late Shift to Positive Nitrogen Balance.*—In the 10 patients who were given no supplementary nitrogen, negative balance persisted until oral feeding was reestablished and the nitrogen intake exceeded 10 gm. in 24 hours (Chart 2). No patient exhibited a positive balance on a smaller intake than this at any time, and in the first postoperative week quantities of nitrogen greater than this were required to reverse the negative nitrogen trend, as is shown in the preceding paragraph. On the second postoperative day, intakes of 13, 17, 19, and 20 gm., although reducing nitrogen loss, failed to prevent a deficit. On Day 7, on the other hand, similar intakes reversed the balance. Intakes of 10-11 gm., insufficient on Day 7, were adequate by Day 9.

3. *Secondary Nitrogen Deficits Accompanying the Development of Complications.*—The development of complications led to secondary nitrogen deficits in three cases. One developed pyloric obstruction and the second both obstruction and a subdiaphragmatic abscess (Chart 3). The third patient, an asthmatic, who was given a high nitrogen intake during the first postoperative week, developed atelectasis of the lower lobe of the left lung on the seventh postoperative day. He became febrile (Temperature 103 F) and faltered in his oral nourishment during the second week. Urinary nitrogen output increased when nitrogen intake fell off, and the patient remained in negative balance from the 8th through the 13th day.

4. *Effect of Caloric Intake on Nitrogen Balance.*—Within the limits of our observations increasing the intake of nonprotein calories had no appreciable affect on the nitrogen deficit, the coefficient of correlation between the two being 0.01 (Table 3). Increasing the intake of protein calories, on the other hand, diminished the nitrogen deficit. As might be expected, the correlation between the nitrogen deficit and protein cal-

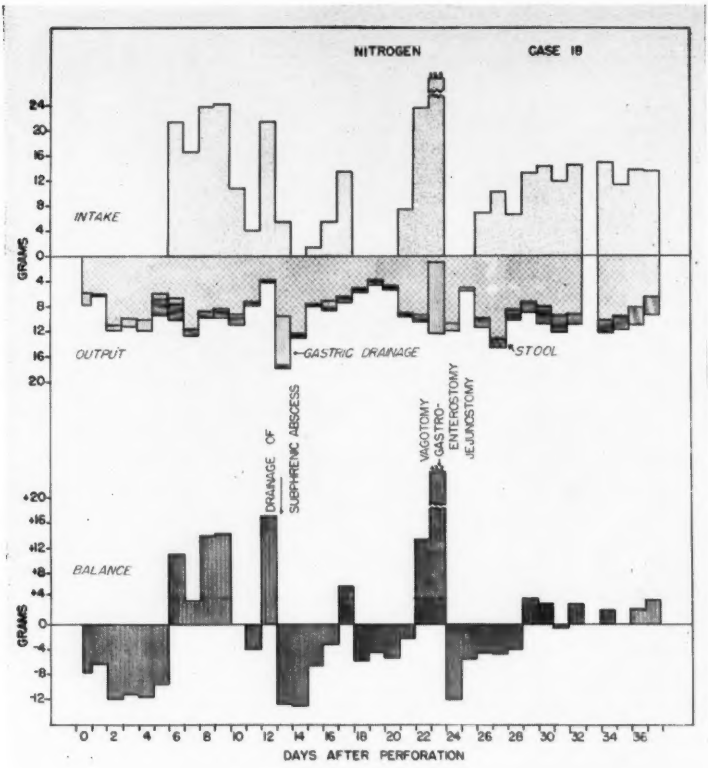


Chart 3 (Case 18).—Secondary nitrogen deficits accompanying the development of complications. A transient increase in urinary nitrogen output occurred after closure of the perforation and each of two succeeding operations for the relief of complications, but inadequate intake rather than excessive output was the major factor in the production of nitrogen depletion.

TABLE 3.—*Correlations of Nitrogen, Potassium, and Phosphorus Deficits During First Week After Perforation*

| Relationship Investigated, All Quantities per Kg. per Day | Correlation Coefficient |
|---|---|
| Nitrogen deficit and intake of nitrogen | —0.80 |
| Nitrogen deficit and intake of calories | —0.50 |
| Nitrogen deficit and intake of nonprotein calories | 0.01 |
| Nitrogen deficit and intake of protein calories | —0.81 |
| Nitrogen deficit and intake of potassium | 0.03 |
| Nitrogen deficit and intake of phosphorus | —0.49 |
| Potassium deficit and intake of potassium | —0.71 |
| Potassium deficit and intake of calories | —0.19 |
| Potassium deficit and intake of protein calories | 0.01 |
| Potassium deficit and intake of nitrogen | 0.02 |
| Potassium deficit and intake of phosphorus | —0.29 |
| Phosphorus deficit and intake of phosphorus | —0.58 |
| Phosphorus deficit and intake of calories | 0.18 |
| Phosphorus deficit and intake of potassium | —0.20 |
| Phosphorus deficit and intake of nitrogen | —0.55 |

ories is identical with the correlation between the nitrogen deficit and nitrogen intake (—0.8).

### Serum Protein Concentration

The concentration of the serum protein was affected by other factors than protein intake and did not follow the balance of nitrogen. In the first paper of this series the observation of high initial serum protein concentrations in these patients was reported.[21] These high levels were considered to represent hemoconcentration, the existence of which was supported by other evidence. The subsequent drop in serum protein concentration coincided with fluid therapy but also occurred during the phase of nitrogen loss just described, continuing after rehydration was complete. The lowest protein

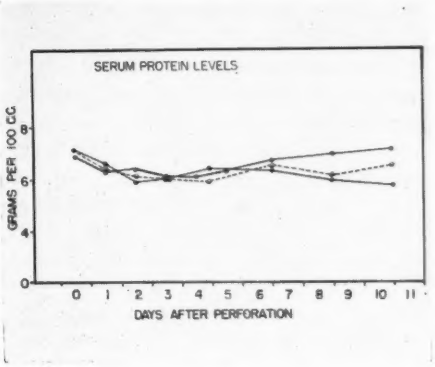


Chart 4.—Elevated initial serum protein concentration and subsequent fall with rehydration and nitrogen depletion. The open circles indicate the protein concentration in patients receiving a low nitrogen intake, and the filled circles indicate those receiving a moderate or high nitrogen intake. In the quantities given, parenteral nitrogen therapy did not prevent the drop in serum protein concentration.

concentrations were observed on the third, fourth, and fifth postoperative days, and thereafter a gradual rise occurred (Chart 4). In three cases the protein concentrations stabilized and began their upward trends while both day-to-day and cumulative nitrogen balances remained negative. Despite institution of a high nitrogen intake in the patient who developed the subdiaphragmatic abscess, restoration of the serum protein concentration to normal was delayed beyond the ninth postoperative day (Case 18).

### Relationship of Nitrogen to Potassium and Phosphorus

The losses and gains of potassium, phosphorus, and nitrogen did not parallel each other within the period of study. The potassium and phosphorus deficits, originally large, diminished and gave way to retention while nitrogen depletion continued (Charts 5 and 6). The details of these changing re-

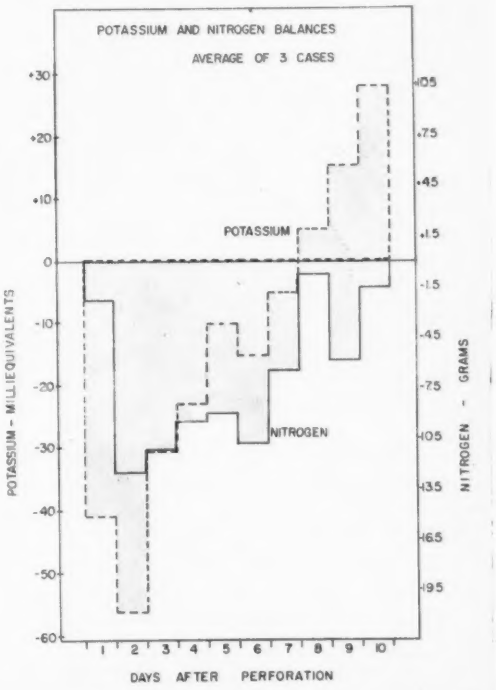


Chart 5.—Comparison of potassium and nitrogen balances. The balances of three cases who received no parenteral nitrogen, potassium, or phosphorus are shown. The scales have been arbitrarily adjusted to the approximate ratio of potassium to nitrogen in lean muscle. A 2.7-mEq. change in potassium balance is equivalent on this chart to a 1-gm. change in the nitrogen balance. The initial potassium loss was great, diminishing steadily after the second postoperative day, in contrast to the more constant nitrogen loss. Increasing the intake of either potassium or nitrogen decreased the respective deficits. The intake ratio of potassium to nitrogen was high on the 8th through the 10th day, when nitrogen depletion and potassium retention occurred simultaneously.

lationships are described in the next two sections.

1. *The Balance K/N Ratios: Relatively Greater Initial Potassium Deficit and Subsequent Greater Loss of Nitrogen.*—On the first postoperative day, the potassium and nitrogen deficits of 12 patients averaged 37 mEq. and 2.9 gm., respectively (K/N ratio: 13 to 1). On the following day both losses increased, but the change in potassium balance was small relative to the increased loss of nitrogen. Forty-three milliequivalents of potassium and 10.1 gm. of nitrogen were lost, reducing the K/N ratio to 4 to 1. Thereafter, the potassium deficit decreased steadily, giving way to potassium retention on the seventh postoperative day. The decrease in nitrogen loss was slower in developing, so that the ratio of potassium loss to nitrogen loss fell steadily, reaching zero on the seventh day.

The patients' average nitrogen balance was negative and the potassium balance positive from the 7th through the 11th postoperative day.

2. *The Balance N/P Ratios: Relatively Greater Initial Phosphorus Loss and Subsequent Greater Nitrogen Deficit.* — Similar changes were observed in the ratios of the nitrogen-phosphorus balances. On Day 1, when the average nitrogen deficit was 2.9 gm., the phosphorus deficit was 0.7 gm. (N/P ratio: 4 to 1). On the succeeding day, the nitrogen deficit increased while the phosphorus deficit decreased, the nitrogen loss being 10.1 gm. and the phosphorus loss 0.5 gm. Accordingly, the N/P ratio rose to 20 to 1. For the remainder of the first postoperative week, the ratio of nitrogen to phosphorus loss was higher than it had been on the first postoperative day. No balance N/P ratio below 13 to 1 was observed.

From the 7th to the 11th postoperative day the average phosphorus balance was positive, but nitrogen depletion continued.

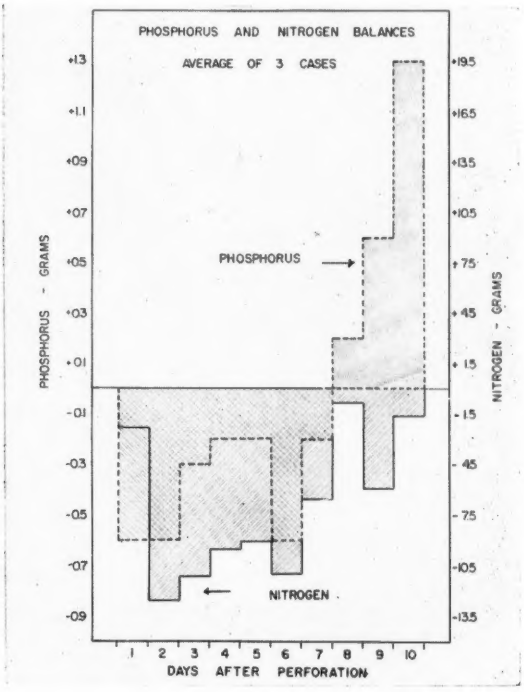


Chart 6.—Comparison of phosphorus and nitrogen balances. The balances of three cases who received no parenteral nitrogen, potassium, or phosphorus are shown. The scales have been arbitrarily adjusted to the approximate ratio of nitrogen to phosphorus in lean muscle. A 15-gm. change in nitrogen balance is equivalent on this chart to a 1-gm. change in phosphorus balance. On the first postoperative day, phosphorus depletion was greater relative to nitrogen depletion than on any subsequent days. Increasing the intake of either nitrogen or phosphorus reduced the respective deficit. The intake ratio of nitrogen to phosphorus was low on the 8th through the 10th postoperative days, when phosphorus retention and nitrogen depletion took place simultaneously.

3. *The Effect of Intake on the Balance Ratios.*—In the second paper of this series it is shown that increasing the intake of potassium or phosphorus decreases the respective deficit, although augmenting the output.[22] In this paper a similar effect of nitrogen therapy is demonstrated. These observations are reflected in the balance K/N and N/P ratios. On the first postoperative day, the intake of nitrogen was high relative to the potassium and phosphorus intakes (K/N ratio: 3 to 1; N/P ratio: 8 to 1) and the nitrogen deficit was low. The nitrogen deficit showed a relative increase, and the phosphorus and potassium deficits decreased to the vanishing point, as the intake K/N ratio rose and the N/P ratio fell.

## COMMENT

With all that has been written about the excessive loss of nitrogen following trauma and operations, the extent of nitrogen loss in the patient with a surgically closed perforated ulcer was a surprise. No more nitrogen was lost than is lost by a normal person on a comparably reduced intake. The reasons for the meager loss of nitrogen in the urine of the ulcer patient during the first postoperative week are not understood. Perhaps the loss is due, as Howard has suggested in his patient who was ill before sustaining a fracture, to the depletion and stress preceding the trauma.[5]

The practical application of this finding is that the nitrogen deficit in these patients is a matter of inadequate intake. The deficit is preventable.

The question of the effect of a nitrogen deficit upon the clinical course of the ulcer patient postoperatively is not readily answered. No measurable evidence of an ill effect of the nitrogen deficit was obtained in the study, but it seems foolish to permit losses of body protein which can be averted. If there is a direct means of maintaining nitrogen balance (by increasing nitrogen intake), it would not make sense to allow an already undernourished patient to become further depleted. Our plan of therapy, therefore, should provide nitrogen.

In management of the nitrogen deficit, emphasis must be placed on intravenous protein therapy. The position of the injury in the stomach or duodenum precludes the use of the gastrointestinal route for several days. Amino-acid hydrolysates are the obvious answer to this need. Their use intravenously is an expensive economy, since much of the injected material is lost through the kidneys, but if enough is given balance can be achieved.

At the start of this study, it was hoped that something could be learned about tissue breakdown and repair in the postoperative patient by comparison of the potassium, phosphorus, and nitrogen balances. Albright, for example, utilizing the ratios of these three tissue components in food and excreta of patients at rest on a metabolic ward, has reconstructed theoretic changes in bone and muscle.[29] In the ulcer patient, however, a similar examination fails to indicate specific tissue changes. Apparently the metabolic turmoil engendered by perforation and its treatment is too complex to yield to such simple analysis. If breakdown and repair of a single tissue were the only process involved, close correlations between the balances of potassium, phosphorus, and nitrogen should be found, but in the patient with a healing ulcer a multiplicity of changes are involved which may affect the metabolism of these three building materials. Involved, among others, are the varying ratios in different tissues ‖ and the diverse roles of the three substances in intermediary metabolism. The fluid shifts accompanying edema and its resorption may affect the electrolytes more than nitrogen; alterations in metabolic rate may affect nitrogen more than the electrolytes. Finally, as has been shown, it is possible to diminish the loss or increase the retention of any one of the three substances independently by altering its intake. It would be oversimplification to try to explain the change in balance ratios solely on the basis of tissue damage and repair.

‖For example, the N/P ratios of skin, muscle, and liver tissue have been reported as 16.6, 17.2, and 10.3 to 1.[30]

The single patient not operated on offers a startling contrast to those treated by operation. His urinary nitrogen excretion, and therefore his nitrogen deficit, was the greatest of all the cases. This is the more surprising, since he was spared the trauma of operation. This patient was not operated upon owing to a mistaken impression that perforation had occurred 24 hours before admission. The patient, a prisoner of low mentality who gave a constantly changing history, was subsequently found to have had his ulcer perforate 11 hours before entry. He was not unusually sick, and the signs of peritonitis were not greater or more prolonged than in the cases with operation. Since this observation of a greater nitrogen loss in the patients not operated on has not been repeated, we hesitate to comment further than to call attention to the difference.

## SUMMARY AND CONCLUSIONS

The nitrogen balance of 17 patients with perforated peptic ulcers was studied. Sixteen patients were treated by surgical closure and one by conservative management.

The loss of nitrogen in the urine of the patient whose ulcer is treated by surgical closure is no greater than that expected in a normal person on a comparably reduced dietary intake. The excessive nitrogen excretion expected after similar trauma in previously healthy persons is not found. The possible relation of this finding to the stress and depletion preceding perforation is discussed.

Inadequate nitrogen intake, rather than excessive excretion, is responsible for the major portion of the postoperative nitrogen deficit in the patient with a perforated ulcer.

The postoperative nitrogen deficit of the perforated-ulcer patient can be reduced by intravenous administration of amino-acid hydrolysates. This in as expensive economy, since much of the injected material is lost through the kidneys.

A distinction is drawn between nitrogen deficit and nitrogen depletion. The reasons for averting unnecessary nitrogen losses are considered.

The serum protein concentration is only a limited indicator of the state of nitrogen depletion or repletion. Similar drops were seen in cases with large and small cumulative nitrogen deficits.

The balances of potassium, phosphorus, and nitrogen do not run parallel to one another. Calculations of their ratios do not yield specific information regarding tissue breakdown and repair. The reasons for this are discussed.

The single patient not operated on, although not sicker than the average, excreted more nitrogen in the urine and built up a larger nitrogen deficit than any of the patients treated by operation.

The details of the plan of nitrogen therapy will be presented in the sixth paper[31] of this series.

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## Case Reports

# FAMILIAL POLYPOSIS OF THE COLON

FELIX C. LEWKOWICZ, M.D., M.Sc.
and
EDWARD G. JOSEPH, M.D., M.S., Jerusalem

HEREDOFAMILIAL diffuse polyposis of the colon presents a clinical entity which differs by its pathological and clinical characteristics from other forms of polyps occurring in the intestinal tract.

Since this is not a common disease, only a small number of cases can be observed by a surgeon during his lifetime. The first description and recognition of the familial predisposition to this disease are accredited to Harrison Cripps, whose classical work was published in 1882. Dukes [10] and Lockhart-Mummery [15] later clarified and stressed its familial tendencies, distinguishing the condition under discussion from the acquired inflammatory type of the disease. They stated that it is a disease transmitted by both sexes and appears to be inheritable according to the Mendelian laws. Unlike similarly inherited conditions, such as hemophilia, familial polyposis is not found when a child is born, but develops at about the age of puberty or even later in life.[4] In this respect it behaves like hereditary optic atrophy.[17] The authors asserted further that the condition is an example of gene mutation, the gene being either dominant or recessive. If the gene is dominant, the disease will be found in every generation, but if the gene is recessive, the disease will occur only when both parents carry the mutated genes. The hereditary transmission of the mutated gene shows no predilection for either sex. The average age of patients showing signs of familial polyposis is between 20 and 30 years, but, occasionally, the disease is seen beyond these age limits.[23]

Submitted for publication March 7, 1955.

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### BENIGN-MALIGNANT POLYP OF THE COLON: CLINICAL AND PATHOLOGICAL FEATURES

Polyposis of the colon should be divided into two different groups: 1. Inflammatory polyposis or pseudopolyposis, as seen in the course of a protracted ulceration of the colon from amebic dysentery or in ulcerative colitis; small islands of mucosa between the ulcers hypertrophy, become fibrotic, and take on the gross appearance of polyps. This is an acquired disease not found among other members of the patient's family. 2. Familial polyposis, in which the disease occurs in two or more members of the family.

The clinical incidence of the first type is high. It is seen in about 30% of persons over the age of 30 years. The percentage may be even higher since many polyps are symptomless. Atwater and Bargen [2] found in 241 consecutive autopsy cases an incidence of 68% of polyps of the colon, as proved by microscopic examination. Acquired polyposis usually manifests itself at the age of 40 and above. The number of polyps in the acquired type, despite its incidence in the older age group, is far less, and the polyps are limited mainly to the rectosigmoid. The familial type, on the other hand, is rare and occurs mostly between the ages of 20 and 30 years.[15]

In a case of familial polyposis the colon is studded with thousands of adenomas and small papillomas of all sizes and shapes, including the flat or pedunculated varieties. Nevertheless, these tend toward greater uni-

formity in size and are scattered throughout the entire colon; only occasionally do segments escape involvement.[12]

Polyposis limited to one part of the colon may become generalized. Sometimes, the polyps are so close together that the normal mucosal pattern is hidden. The diseased bowel is uniformly larger than the normal; the haustral markings persist; the adjacent mesentery is usually thickened and edematous, with several enlarged lymph nodes. In addition, the colon is heavy and fleshy on palpation and does not collapse on pressure, while its weight is greatly increased. Malignant polyps appear, which are broad based, invasive of the wall, and diffusely indurated. Microscopically, the structures vary considerably. The polyp has a stalk-like connective tissue central structure infiltrated with lymphocytes and covered by a hyperplastic columnar epithelium. Mitotic figures are numerous, and the cells appear to infiltrate the supporting membrane. There exists an intrinsic urge of the epithelium to proliferate. All these lesions eventually become malignant, and typical hyperplasia is frequently to be found in them. As the process progresses toward malignant neoplasia, the cells become more characteristic and invade the deeper layers of the connective tissue center. In evaluating the progress made by a polyp toward carcinoma, therefore, it is important to examine the stalk of the polyp and to determine whether the base and underlying bowel have been invaded.[12]

All observers are aware of the intimate relationship between simple adenomatous polyps and carcinomas of the colon and rectum. Swinton [23] found that 14% of carcinomas of the colon arise from benign polyps and demonstrated this fact by microscopic studies. Buie [6] showed that only 4.6% of patients with solitary polyps of the rectum subsequently developed carcinoma. On the other hand, Morton [18] felt that the greatest part of the polypoid lesions of the colon-rectum would become malignant if allowed to remain *in situ*. They are, therefore, true tumors and must be clinically considered as precancerous lesions.

Regarding the heredofamilial type of polyposis, most authors agree that the development toward carcinoma occurs in a very high proportion of cases. The malignant potentialities of each individual polyp, when multiplied by the myriads of polyps present, make this disease the most dangerous precursor of carcinoma known. It is interesting to note that most of the living nontreated patients with familial polyposis are under the age of 30. Those over 30, on the other hand, die or are known to have carcinomas. It becomes apparent that the closer the patient is to the age of 30 before therapy is instituted, the greater is the possibility of malignant disease developing, and the smaller are his chances of cure. Schaffer [22] points out that the average duration of symptoms is three to four years.

No single symptom or symptom-complex can be considered pathognomonic of diffuse familial polyposis. In some cases bleeding and diarrhea may occur. Some patients complain of periodic gripping pains in the lower abdomen together with tenesmus. The stool appears with blood streaks and mucus. Patients complain of general weakness. In persons with the disease, the physical findings are within normal limits. The only suggestive sign is discovered on digital examination of the rectum. A subsequent rectosigmoidoscopic examination is of great diagnostic value. The lesions are thus visualized, and tissue can be removed with a biopsy forceps from suspicious areas for histologic examination. Of equal importance as a diagnostic procedure is roentgenologic examination of the colon. By means of the double-contrast method, as modified by Weber,[25] these polypoid lesions can be well demonstrated. The last two examinations may confirm the diagnosis with a 100% certainty.

## REPORT OF CASES

CASE 1.—A 48-year-old woman was admitted to our surgical department because of colicky pains

in her abdomen. She was the mother of five healthy children. Her sixth child, a daughter, had the same condition (Case 2). Fifteen years ago, the patient's older brother began to complain of bloody stools. He was examined in another hospital, and an operation on the colon was performed. Several days later, while still in the hospital, he died. A second brother, who underwent an operation on the rectum at the age of 40, because of bleeding from the anus, died only a few years ago at the age of 68.

A year and a half before coming to us, the patient had been operated on in another hospital, where an ileotransversostomy was performed because of stenosis due to a tumor in the hepatic flexure. Since the operation, she had been feeling well and was free from pain. During the past few months, however, she compalined of general weakness, loss of weight (about 15 kg. in the last two and a half months), and attacks of abdominal cramping, with diarrheal stools three times daily. No blood was ever found in her stools. Her loss of appetitie was not accompanied by vomiting. On examination, we found the patient to be in good general condition without any abnormal physical findings, except for a mass the size of an orange palpable in the right upper abdomen under the liver. Her blood pressure was 170/125. A barium enema was administered which showed the original stenosing tumor in the hepatic flexure. The lesion had not been removed in the preceding operation because of suspicion of ameboma, as the patient had amebas in her stool. After she left our hospital, intensive medical treatment was prescribed for the patient, and she was advised to return in a short time for reexamination prior to a radical operation. However, because of her daughter's illness, she postponed her readmission until her own condition became more aggravated. On rectoscopic examination, a sessile polyp of the size of a cherry was removed 15 cm. from the anus. The histologic examination of the polyp showed possible cancer. On operation, a partial colectomy was performed, and the tumor was excised together with the adherent tissues (lower part of the right liver lobe and a part of the anterior abdominal wall). After the operation, the patient recovered uneventfully for six months. She then started to complain again of abdominal pains and constipation.

Her daughter, in the meantime, also complained of abdominal pain. She was examined, and diffuse polyposis was found. A suspicion was aroused of familial polyposis with malignant degeneration in the mother, and, consequently, the mother was reexamined rectoscopically. The polyp in the sigmoid recurred; it increased markedly in size, and on palpation a large mass was felt in the lower abdomen. A laparotomy was performed, with the aim of removing the entire diseased colon, but a large left ovarian tumor infiltrating into the uterus and pouch of Douglas was revealed. Since a more radical operation was impossible, only an excision of both ovaries and the uterus was done. The microscopic examination revealed an adenocarcinoma mucoides infiltrating the left ovary, adnexa, and uterus, probably metastatic to the intestinal neoplasm. The patient was then released and was sent to continue treatment with radiation. Eight months after the operation, the patient died of cachexia. No postmortem examination was performed.

Case 2.—The 18-year-old daughter of the patient in Case 1 complained of periodic gripping pains in the lower abdomen and of diarrhea. She had had these disturbances from the age of 10 years, when she first noticed blood streaks in her stool. A proctologic examination performed by us revealed multiple polyps in the rectum and colon. A biopsy specimen from the rectum showed adenomatoid polyp. An x-ray done by means of the double-contrast method gave the typical picture of diffuse polyposis of the colon. Results of physical examination were normal. No anemia was found. Since the polyps were located very low in the rectum and were sessile-broad in form, with a tendency to rapid multiplication, and since new polyps appeared one month later in place of those removed for biopsy, it was decided to remove the entire colon and to perform an ileostomy. This was successfully done. Five years have passed since the operation, and the patient is free of complaints. She feels well, has gained weight, and uses the Rutzen bag for her ileostomy. A year ago she gave birth to a healthy child.

## COMMENT

Diffuse polyposis of the colon should be considered a malignant or potentially malignant condition. Once the diagnosis has been established, the disease should be treated radically. The program for treatment must, therefore, be definite: early extirpation of the whole polyp-bearing colon. There are two methods for the surgical treatment of heredofamilial polyposis:

1. Total colectomy with perineal excision of the rectum, leaving an abdominal ileostomy.[8] Most authors do not suggest the removal of the rectum and the establishment of a permanent ileostomy unless a carcinoma has appeared in the rectal or sigmoid segment. Ravitch and Sabiston, in 1947,* proposed a different treatment for patients re-

* References 19 and 20.

quiring total colectomy for benign colonic lesions, e. g., ulcerative colitis or familial polyposis. They suggested a pulling-through of the ileum and its attachment to the anal skin. This procedure leaves the anal sphincter unimpaired.

2. Fulguration of the rectal polyps, followed a month later by the performance of a one-stage subtotal colectomy with an end-to-end anastomosis of the terminal ileum to the distal 20 cm. of the rectum.[15]

The factors which determine the choice of the second operation are the following: 1. There must be no carcinoma present in the rectum at the beginning of treatment. 2. The rectal segment remaining after surgery must be amenable to complete visualization with a proctoscope. 3. This segment must be completely cleared of polyps. 4. Every three months for the remainder of the patient's life an examination of the rectal segment together with biopsy and destruction of new polyps (if any) must be performed. This set of conditions should be satisfied prior to adopting the operation with retention of the rectal segment.† The advantage of this procedure is obvious, especially when young persons are concerned, as it permits them to carry on a normal and active life. The inconvenience of routine rectoscopic examination is negligible as compared with that of an ileostomy. The only criticism of this procedure, which purposely leaves behind a polyp-bearing organ, may be directed from the standpoint of the surgical rules dealing with potentially malignant processes. Nevertheless, the possibility of a constant and careful follow-up control of familial polyposis may lessen its dangers and makes this procedure an ideal operation for it.

SUMMARY

Familial polyposis is a rare inheritable disease transmitted as a Mendelian-dominant characteristic. No pathognomonic symptoms and no definite physical findings make the disease distinguishable in its early and operable stages. Clinical and pathological differences between familial and acquired polyposis are noted.

The accepted method of treatment of familial polyposis without carcinoma in the rectum is an end-to-end anastomosis of the terminal ileum to the rectum, thus preserving the anal sphincter.

A family history with a five-year survival in one case is reported, with the view of adding to the number of cases recorded in the literature.

† References 1, 4, 5, 14, and 22.

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# Surgery Illustrated

## A Modified Halsted Inguinal Herniotomy Aimed at Preventing Recurrence

ROBERT ELMAN, M.D., St. Louis

Despite the fact that the radical treatment of inguinal hernia has been known and practiced for more than 65 years, there is a general belief that the recurrence rate is still considerable. Perhaps the most interesting evidence thereof was reported in 1920 by Taylor,[1] who found in an analysis of 910 cases that whereas the average rate of recurrence was 5.6% for indirect and 18.0% for direct hernia the record for each individual surgeon varied, the highest being 28.5% and 30.0%, respectively. This study shows that there is need for greater scrutiny into the technique of the procedure in order that all surgeons may enjoy relative freedom from recurrence. Regardless of details, one of the basic principles (after high ligation of the sac) is the need during repair for careful suture of fascia to fascia *without tension.*

The technique described herein has been used by me in a series of 40 cases of both direct and indirect hernias operated upon personally in private practice without a single known case of recurrence, based upon a follow-up of three years or more. All patients of course were adults, but it was employed in several with recurrent hernia and in a number of old obese patients with large defects and poorly developed fascia. While it is true that the procedure used may not be necessary in every instance, my own experience would suggest that it is a successful and relatively simple insurance against recurrence.

The two modifications in the original Halsted technique herein represented are not new. The use of autogenous fascia obtained from the external oblique was described[2] by McArthur in 1901. Actually, he used two strips of fascia. After a follow-up of three years or more, he reported[3] on 93 cases operated upon by several surgeons in

Recorded for publication Dec. 10, 1955.

From the Department of Surgery, Washington University and the Barnes Hospital.

The illustrations were executed by Mr. W. Howard French, aided by preliminary sketches by Dr. Harry Crawford.

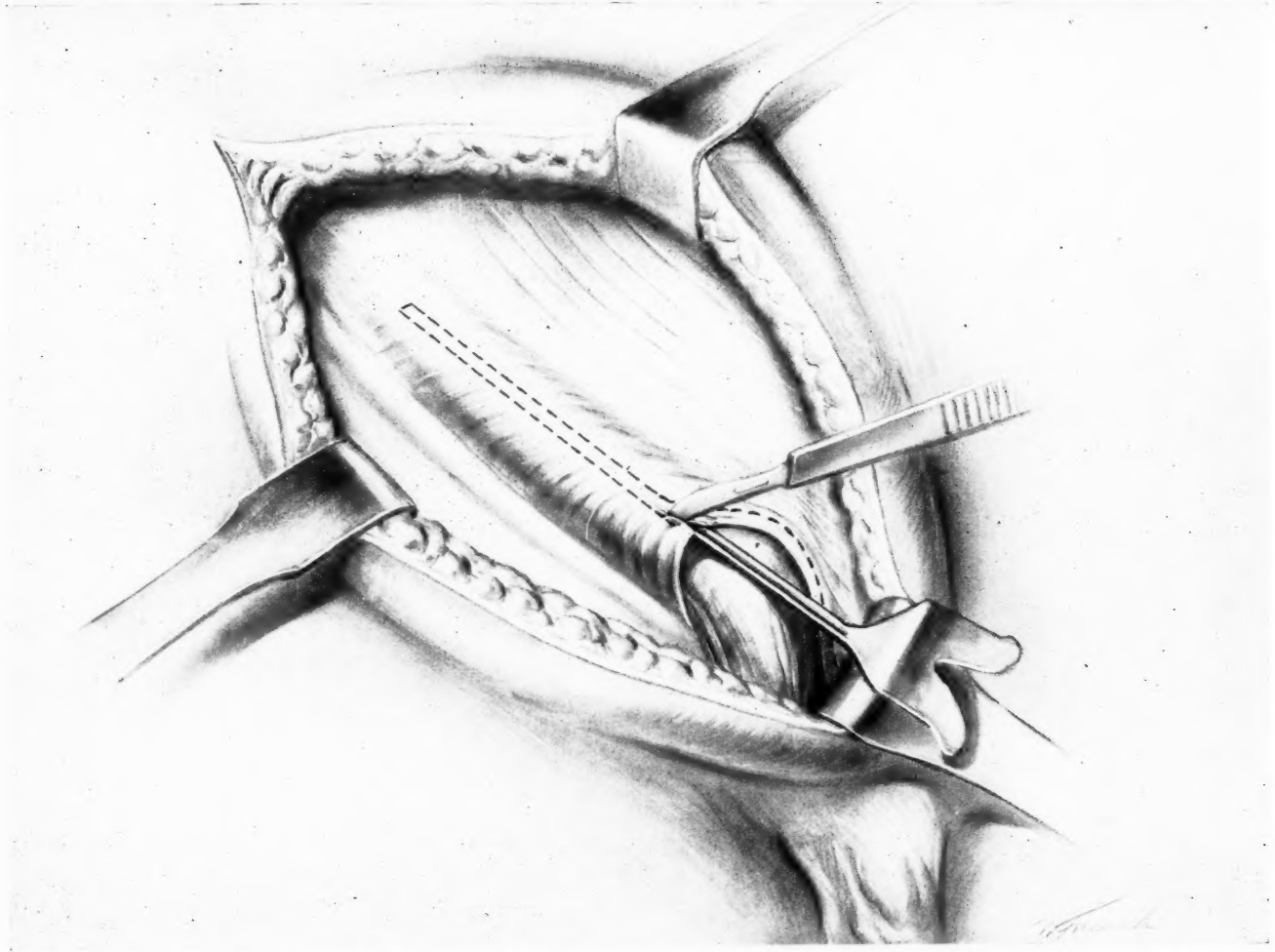

Fig. 1.—As the inguinal canal is opened a strip of fascia from the external oblique is removed, including the upper edge of the subcutaneous ring down to its attachment at the symphysis, as illustrated by the dotted lines. The free end is then fixed to a Gallie needle in the usual way.

Fig. 1.—As the inguinal canal is opened a strip of fascia from the external oblique is removed, including the upper edge of the subcutaneous ring down to its attachment at the symphysis, as illustrated by the dotted lines. The free end is then fixed to a Gallie needle in the usual way.

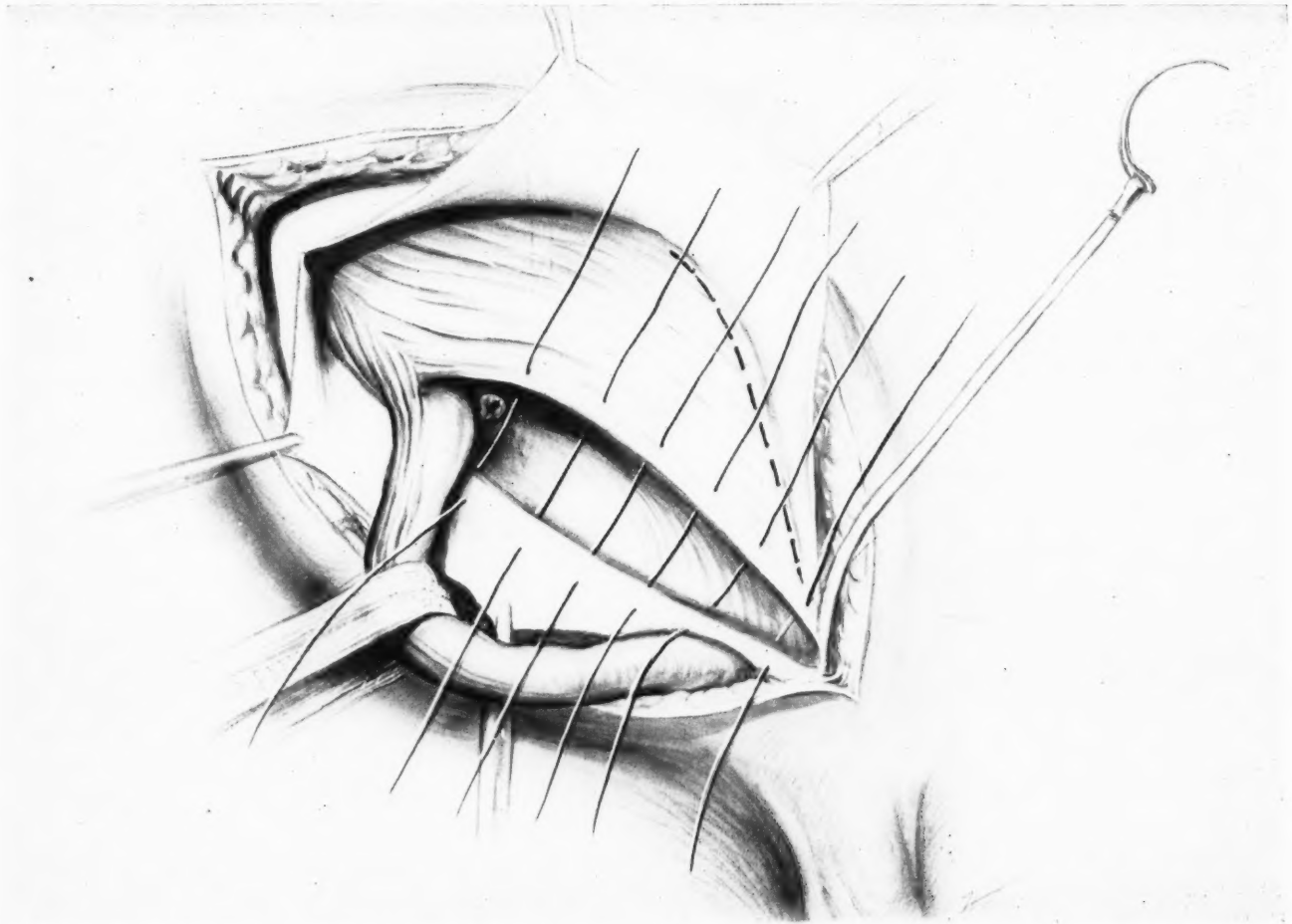

Fig. 2.—The sac has been identified, separated from the cord, ligated high, and excised. The cord is carefully mobilized and detached from its bed and transplanted high up into the belly of the internal oblique. The external oblique has been mobilized as high as possible. The dotted line shows the relaxing incision made in the internal oblique fascia just below its attachment with the external oblique and the anterior rectus fascia. Silk sutures are placed as usual but are not tied until later as the fascial strip is being placed.

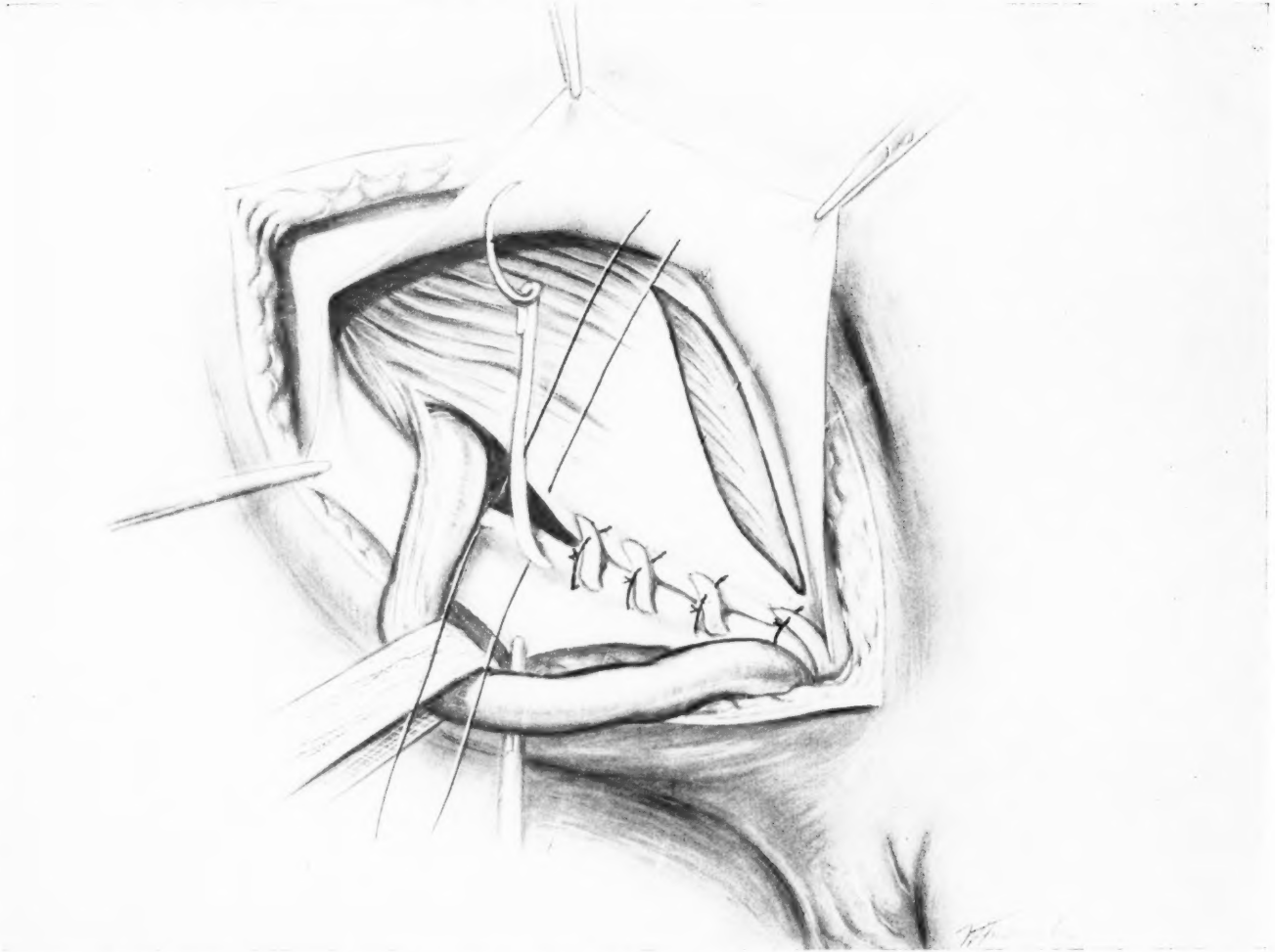

Fig. 3.—As the fascial strip approximates Poupart's ligament with the internal oblique, each silk suture is passed through it and tied, thus fixing the fascia in situ at this point. Not shown is the usual method of staggering the insertion of each silk suture (and fascial strip) into Poupart's ligament.

Fig. 3.—As the fascial strip approximates Poupart's ligament with the internal oblique, each silk suture is passed through it and tied, thus fixing the fascia in situ at this point. Not shown is the usual method of staggering the insertion of each silk suture (and fascial strip) into Poupart's ligament.

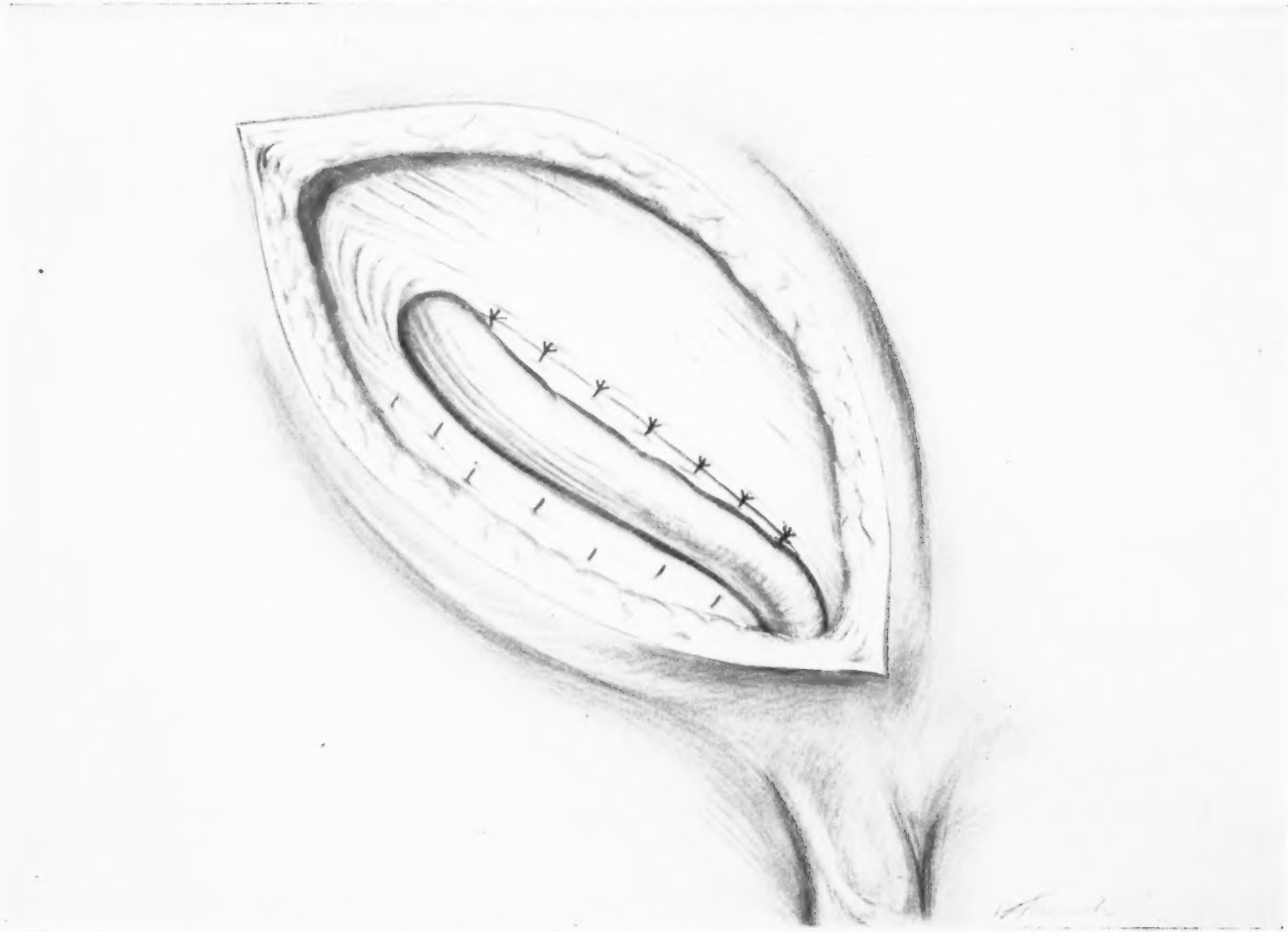

Fig. 4.—The external oblique is approximated beneath the cord, thus completing the operation. Not shown is the fascial suture between the lower row of silk sutures visible through Poupart's ligament.

Before closure, the wound is thoroughly but gently irrigated with isotonic saline or Ringer's solution to remove loose fat and other tissue debris, a detail which I have found insures a clean, dry wound and minimizes the likelihood of superficial infection.

this manner without a single recurrence. Although this technique is mentioned in most monographs on hernia, it is seldom described in detail and it seems to have been forgotten or given up by most surgeons. The second modification is the relaxing incision high up on the internal oblique fascia. This practically always permits this fascia to be sutured to Poupart's ligament without tension, even in the face of large defects in the inguinal canal. Although it was probably originally mentioned by W. S. Halsted, it was described [4] by W. F. Reinhoff in 1940.

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*Scientific Exhibit*

# PERCUTANEOUS ANTEGRADE PYELOGRAPHY & TRANSLUMBAR NEEDLE NEPHROSTOMY IN HYDRONEPHROSIS

by WILLARD E. GOODWIN and WILLIAM C. CASEY
Los Angeles

THIS EXHIBIT presents two techniques and demonstrates examples of their usefulness in diagnosis and treatment of hydronephrosis. Both are performed with local anesthesia and roentgenographic control.

Antegrade pyelography [1] consists of pyelograms made after lumbar needle puncture of a hydronephrosis. Urine is aspirated through a 6 in. 18- or 19-gauge spinal needle, and urographic contrast medium is injected to outline the renal pelvis and ureter to the point of obstruction. It should be reserved for cases of large hydronephrosis in which the diagnosis cannot be made by conventional techniques.

Trocar nephrostomy [2] (or pyelostomy), a logical sequel to this technique, consists of lumbar tap of a large hydronephrosis with a 12-gauge thin-walled needle through which polyethylene tubing is inserted to afford temporary urinary diversion. It should be reserved for large hydronephroses in which temporary drainage is desired without operative intervention before later definitive treatment.

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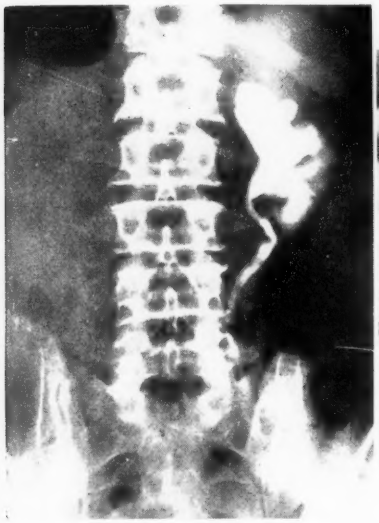
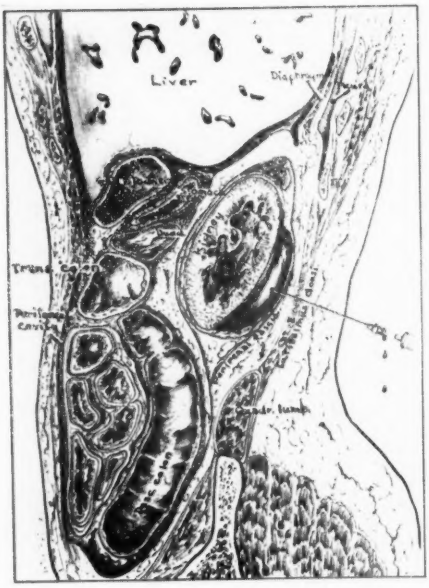
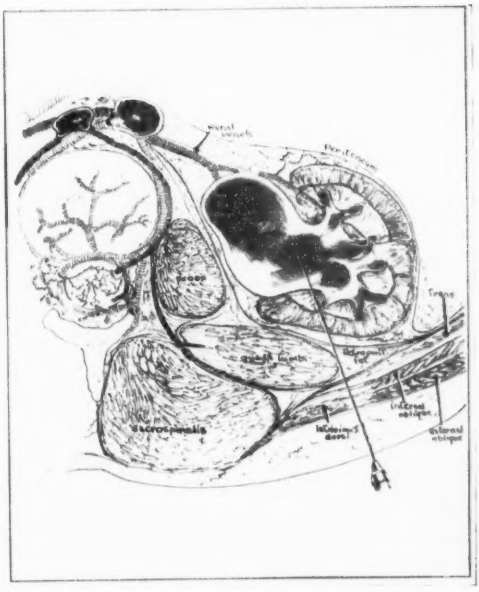
A portion of this work was supported by a grant from the Schering Corporation.

From the Division of Urology, The University of California Medical Center; Veterans Administration Center; and The Harbor General Hospital.

# TRANSLUMBAR NEEDLE NEPHROSTOMY IN HYDRONEPHROSIS





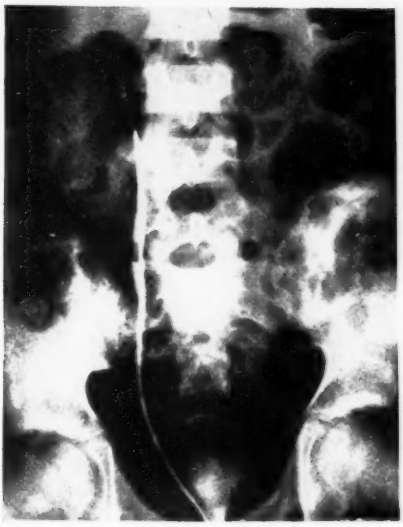




1. **TECHNIQUE:** A lumbar tap of the hydronephrosis is performed with a #12 or #14 gauge 6 inch needle. Plastic tubing is passed through the needle and left in place.

2. This has been used nine times in seven patients. Satisfactory drainage from one day to 3½ months.

3. It should be reserved for use in large hydronephroses when temporary urinary diversion is desired before definitive surgery.

**SURGICAL TRAUMA URETEROPELVIC JUNCTION**



CARCINOMA OF THE URETER





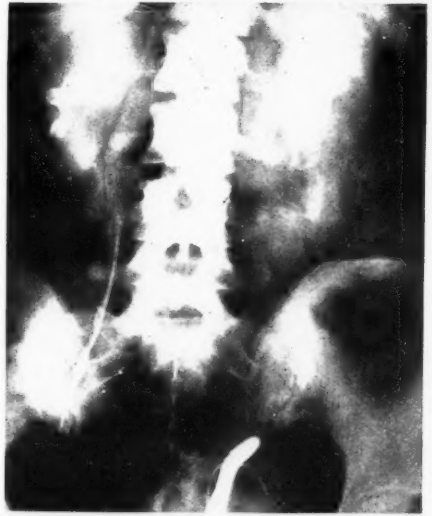


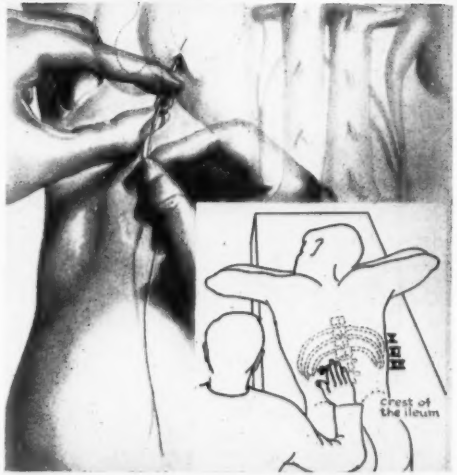


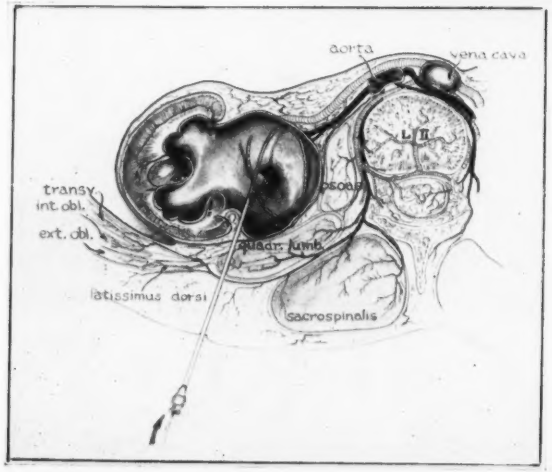


LOWER URETERAL OBSTRUCTION AFTER HYSTERECTOMY

LOWER URETERAL OBSTRUCTION AFTER HYSTERECTOMY





URETEROSIGMOIDOSTOMY







Exhibit Prepared by
MEDICAL ILLUSTRATION LABORATORY
VETERANS ADMINISTRATION CENTER
LOS ANGELES
Drawings by
MARY P. GOODWIN

from the DIVISION OF UROLOGY
- University of California Medical Center
- The Veterans Administration Center
- The Los Angeles County Harbor General Hosp.

LOS ANGELES

## ANTEGRADE PYELOGRAPHY IN HYDRONEPHROSIS

**DEFINITION:** Pyelograms may be made by simple translumbar needle puncture of the renal pelvis and injection of urographic material after aspiration of urine in cases of hydronephrosis. This has been called "antegrade pyelography."

**TECHNIQUE:** Number 18 or 19 gauge spinal needles are used after local anesthesia with procaine.

Urine is aspirated for culture and microscopic study.

A urographic contrast medium (neo-iopax or urokon 30% to 75%) is injected with barbotage to fill the hydronephrosis.

**CAUTION:** the volume of contrast medium injected should always be less than the amount of urine aspirated.

# THE LITERATURE

February Fifth
1 9 5 4

RENAL "TAPPING"

Urologists' Correspondence Club

Dear Fellow Members:

The year 1954 began with hope and expectancy, but a terrific shock occurred to us in January that made our hearts shudder. Jimmie Lee Pittman, my associate and a fellow member of this club, died suddenly and along with it brought an end to a harmonious partnership of more than twenty-five years.

To practice congenially together in this modern life requires, of course, a certain amount of ability, but along with it one must have tolerance and an understanding of each other's faults. The latter I'm sure we had. He will surely be missed.

Well, the whip is cracking over my head, so here goes my contribution:

One cannot be overstocked with diagnostic procedures in dealing with the various kidney problems, although there are some that should be retired. One simple procedure that is not used often enough nowadays but which was found of considerable help to us recently, was that of "tapping" a large hydronephrosis, in which case, both excretory and retrograde pyelography got us nowhere.

A female, aged 43, whose present illness started twenty-four hours prior to admission to the hospital began with a dull left flank pain which required no opiates, and followed by anuria the following day with subsiding of pain.

Her previous history was not significant although she gave a vague history of some left renal pathology eighteen years prior to this time; however, this subsided and, with the exception of one episode of very mild hematuria, she had had no other definite symptoms.

Routine physical examination revealed a normal temperature, pulse 110, blood pressure 190/110. Positive findings included a slightly enlarged heart and a palpable mass in the right flank which moved with respiration.

Her initial laboratory study showed a hemoglobin of 7.1 gm. with red count of 3.4 million. White count was 11,300 with slight shift to the right. Her electrolytes were thrown out of balance and at the time of admission N.P.N. was 61 mg. percent and Creatinine 5.4 mg. percent.

At this time we suspected that we were dealing with an oliguria due to a lower nephron disturbance in a patient with an undiagnosed mass in the right upper abdomen.

On cystoscopic examination the bladder was empty with the exception of a few ccs. of almost pure blood. A catheter was passed up the left ureter to the region of the pelvis, but we had no success in passing a catheter up the right ureter, there being complete obstruction just within the orifice. Pyelograms were attempted but were unsuccessful. None went up the right side and apparently there was an obstruction in the upper ureter on the left by which the media would not pass. Following this procedure the catheter was left in place on the left side and in forty-eight hours the urine output became adequate.

P.S.P. was carried out but the function was too poor to calculate.

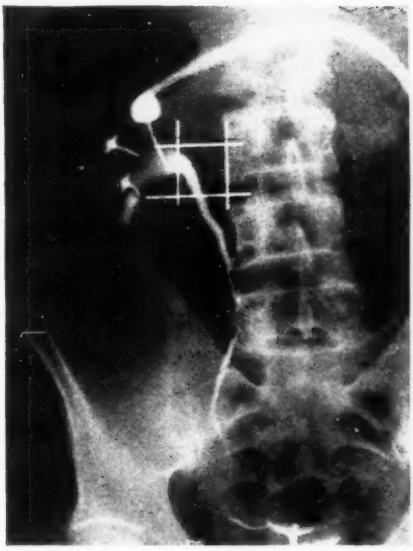
One week later the patient was recystoscoped and a normal left pyelogram was made, and at this time a small calculus was discernible in the lower calyx of the left kidney. Again we were unable to pass a catheter up the right side, but a pyelogram was made by passing a long, 18 gauge needle through the flank into the previously mentioned mass in that area. A successful trans-lumbar pyelogram was made showing a large hydronephrotic sac. We removed the right kidney after a few transfusions and she was discharged seemingly all right, but still carrying a slightly elevated N.P.N.

We may assume that the left renal calculus was responsible for the initial oliguria and hematuria by blocking the left upper ureter. The point that I would like to emphasize in this letter is the use of a trans-lumbar tap in the diagnosis of a renal mass where other methods of pyelography are technically impossible. This method, although used in the past, but never very popular, was of considerable help to us in diagnosing this case and I pass it on to you in the hope it may be of some assistance in some similar case.

Fraternally yours,

Atlanta, Georgia — Earl Floyd, M. D.

Most recently mentioned in Urologist's Correspondence Club Letter



**RESULTS OF THIS STUDY:**

47 CASES
12 *FAILED*... 35 *SUCCESSES*

**REACTIONS:** (Associated with overdistention or extravasation)

- *NAUSEA AND VOMITING*
- *PAIN*
- *FEVER*

(Occurred 6 times in 47 patients)

**CONCLUSIONS:**

1. Antegrade pyelography is a safe and useful adjunct to other accepted methods of urography in selected cases of hydronephrosis.
2. Its use should be reserved for patients with large hydronephroses in whom accurate roentgenographic diagnosis cannot be made by intravenous or retrograde pyelography. It may have special application in children and adults with poorly functioning uninfected hydroneprosis when the urologist wishes to minimize chances of infection and still secure good radiographic studies before surgery.
3. It does not supplant but rather augments conventional methods.

## Special Article

# SPHINCTEROTOMY AND SPLANCHNICECTOMY IN THE TREATMENT OF CHRONIC RELAPSING PANCREATITIS

**PIERRE MALLET-GUY, M.D., F.A.C.S. (Hon.), Lyon, France**

FOREWORD

A sectional meeting of the American College of Surgeons was held in London in May, 1954. Many of the members attending the London meeting also had the pleasure of visiting surgical clinics in Paris, Lyon, and Strasbourg, France. We were very much interested in the surgical work in all the cities and arranged with many of these surgeons to send some of their scientific contributions to this journal from time to time. In this issue Professor Mallet-Guy, of Lyon, is reporting his experiences with sphincterotomy or splanchnicectomy in the treatment of chronic relapsing pancreatitis, a field of surgical research in which he has had considerable experience.

WALTMAN WALTERS, M.D.

In 1913, Archibald, supposing that the origin of every sort of pancreatitis resides in a spasm of the sphincter of Oddi, established the principle of systematic transduodenal sphincterotomy. He obtained a cure in six of eight patients treated according to this plan.

Thirty-five years later, in 1948, Doubilet and Mulholland revived the interest in this problem. In the course of operation they carried out cholangiographic studies preceded by an injection of morphine or an intraduodenal instillation of an acid solution (N/10 HCl) in order to produce artificially a spasm of the sphincter of Oddi. They assumed that every regurgitation in the pancreatic duct produced by this means was an indication for sphincterotomy.

They presented at that time a report on 21 cases of sphincterotomy, with 1 death, 6 improvements, and 14 cures, although most of the favorable results were not permanent.

In 1950, they discussed the same problem again, presenting 73 cases, among which 4 cases of chronic calcified pancreatitis were cured (3 of them after two years). Dragstedt and Elman at the same time also reported a few satisfactory results. Subsequently Jones and Smith published 18 cases without any follow-up; Major and Openheimer reported 23 cases, including satisfactory results in 20 cases and recurrence of symptoms in 3 (12 of these cases, however, had not been followed for more than a year). Gillette reported on 5 cases, of which 4 were cured and one was a failure.

Priestley, Taylor, and Rogers presented 15 cases, of which 2 were failures, 7 had excellent results, and 5 were partially improved. They cited a personal communication of Doubilet and Mulholland, who had operated on 190 cases, of which 9 died, 7 had recurrence, and 173 presented good results.

Thus, after the "boom" of pancreatectomy, followed by the "boom" of sympathectomy, we are witnessing nowadays in the surgical literature the "boom" of sphincterotomy. The only nonenthusiastic note is actually that of Cattell and Warren, who in eight cases of sphincterotomy reported only four satisfactory results. Two of these were cases

Submitted for publication Sept. 17, 1955.

From the Department of Surgery, University of Lyon.

of pancreatic lithiasis, and the others were mostly cases with short histories.

It is not unusual in cases of chronic pancreatitis to notice an associated dystonicity of the sphincter of Oddi, identified by preoperative manometric and roentgenologic studies of the biliary passages. In 1946, together with R. Jeanjean, I had noted 31 cases of hypotonicity and 14 cases of coexisting hypertonicity of the sphincter. In both instances a picture of cholangiographic regurgitation in Wirsung's duct was frequently noticed. Thus, at first sight, I should agree with Doubilet and Mulholland.

However, it seems to me unwarranted to accept a positive response to injection of morphine or instillation of hydrochloric acid as an indication for sphincterotomy. Every functionally normal sphincter of Oddi will react to such a stimulus, with a spasm.

Sphincterotomy, in our opinion, should be reserved for authentic cases of hypertonicity of the sphincter of Oddi. This procedure should not be carried out routinely, especially since hypotonicity appears definitely more frequently at the inception of chronic relapsing pancreatitis.

On the other hand, investigations carried out with M Gangolphe and J. Féroldi, in which the sphincter was sectioned, have enabled us to prove that sphincteric hypotonicity was sufficient to cause the appearance of pancreatic lesions. These lesions are characterized by periductal sclerosis and fatty vacuolation of the parenchyma and duplicating very exactly the lesions caused by regurgitation which we have encountered in man.

It seems to us that indications for sphincterotomy in the treatment of chronic relapsing pancreatitis should be brought back to their proper place and operations on the nervous system particularly should be given the place they deserve.

In fact, surgery of chronic relapsing pancreatitis appears to have plunged into extreme confusion in the past 10 years.

The merits of operations for biliary derivation and of direct surgical intervention on the inflamed pancreatic gland have been successively pointed out. Subtotal gastrectomy, sympathectomy, and sphincterotomy have been successively advocated with the same enthusiasm.

Very often the physiopathologic bases of the procedure are uncertain, the interpretation of the mechanism of the procedure is made a priori, and results often lack follow-up, although the conclusions derived therefrom have not lost any of their value.

Having had a vast experience in the treatment of this disease (my first pancreatectomy for chronic relapsing pancreatitis was performed in 1934, and my first splanchnicectomy in 1942) I have acquired some very specific ideas from the fascinating analysis of the mechanisms of the disease, as revealed by histopathologic and experimental studies. These ideas, I believe, have allowed me to understand the contradictions encountered in the present surgical literature.

The present analysis will be limited to syndromes consisting of a succession of acute and subacute painful episodes expressing successive, severe or mild, episodes of autodigestion leaving behind new areas of sclerosis, constantly more infiltrating, more inflammatory, sclerosis in which future recurrences find their origin.

The clinical description of the disease that I gave in several papers published between 1934 and 1938 is very similar to that given by Comfort, Gambill, and Baggenstoss in 1946.

Let us say that the diagnosis is relatively easy and accurate, provided that, forgetting the fallacious lead of laboratory tests, one seeks the necessary assumptions in the clinical history, the physical examination, and roentgenologic studies, and confirmation in a complete and systematic surgical exploration.

Surgical exploration includes, necessarily, examination of the entire pancreas, where lesions can be different from one point to the other, here clearly inflammatory, there more sclerotic and less active, although

presenting waxy scars, elsewhere at the limit of sclerosis. A biopsy and, if possible, frozen sections will be of great help.

Surgical exploration will also necessarily include examination of the gall bladder and of the common duct, the latter being submitted to manometric and roentgenologic studies.

The indications for a bypass operation on the biliary system in the treatment of chronic relapsing pancreatitis can then be discussed. This can be only a palliative procedure to decompress the common duct and is not a treatment of the disease itself, which is not related to the biliary pathology.

The first attempts at a direct, curative treatment of chronic relapsing pancreatitis were all directed toward resection of the gland. I was led to proceed in this way in September, 1934, on a 55-year-old man who had previously undergone two emergency laparotomies. A left pancreatectomy was performed, and the permanent cure obtained thereby has been confirmed after 18 years follow-up. In this case everything happened as though the various foci of sclerosis left by successive acute episodes had been at the origin of abnormal vasomotor reflexes causing the appearance of subsequent acute episodes in various areas of the gland.

I have performed a total of 14 left pancreatectomies for chronic relapsing pancreatitis, with 2 deaths, 2 recurrences, and 10 excellent results followed, respectively, for 14 to 18 years (4 cases), 4 years (3 cases), and 32 to 40 months (3 cases).

Several publications by Leriche, Soupault, Brocq, Devis and Storck, Carcassonne, Nuboer, and Hershey have confirmed the value of a left pancreatectomy under similar circumstances, whereas, on the other hand, total duodenopancreatectomies were performed subsequently for cases of "pancreatic fibrosis with calcareous deposits," following T. Clagett and A. O. Whipple's suggestions. It appears nowadays that the mutilation of the latter procedure is not in proportion with the inflammatory characteristics of the pancreatic lesions.

Operative procedures on the sympathetic nerves can be included in the same physiopathologic group as left pancreatectomies. This is to say that interruption of the reflex arc responsible for subsequent recurrences can be achieved by a splanchnic block or, even better, by a left splanchnicectomy, whose efficacy I have been able to appreciate ever since 1942. This is not an empirical operation.

Histologic examination of operative specimens following pancreatectomy had shown the frequent incidence of nervous lesions in the inflamed parenchyma.

On the other hand, the principles of left splanchnicectomy rest upon precise experimental bases. Laporte, Compagnon, and du Buit, continuing the experiments carried out by Reilly in 1937, had demonstrated the contingent appearance of acute necrosis of the pancreas among other foci of infarction, following the introduction of foreign bodies in the cervical and thoracic sympathetic ganglia. Subsequently, and after the first clinical results confirming the remarkable efficacy of left splanchnicectomy, I have tried, conversely, to reproduce experimentally the various stages of pancreatitis by stimulating the left splanchnic nerve. Thus, and in association with R. Jeanjean and J. Féroldi, and subsequently with J. Féroldi and E. Reboul, we were able to obtain, successively, lesions of edematous and hemorrhagic pancreatitis. Only excitation of the whole nerve or of its distal end are effective; excitation of the central portion of the left splanchnic nerve or of the right splanchnic nerve are ineffective.

These experimental data will allow us to understand the unfavorable impression often given at the present time by the results of operations on the sympathetic nervous system in the treatment of chronic relapsing pancreatitis.

De Takats and Walter, who introduced this procedure in the United States, in 1947,

resected, in addition to the splanchnic nerve, part of the left thoracic sympathetic chain, although only between the 9th and 12th ganglia. The operation was often carried out on the right side or bilaterally and involved mostly an extensive thoracolumbar sympathetic resection.

Furthermore, sympathectomies are usually carried out by neurosurgeons who frequently lack the operative experience required to determine the nature of the pancreatic lesions; or, in other instances, a laparotomy revealing the existence of a chronic pancreatitis had been performed long before by another surgeon.

Twenty-nine such observations were published between 1948 and 1951. Unfortunately, and in view of the variety of the operative techniques used, it is impossible to interpret accurately the results obtained. These are often excellent. However, de Takats, Walter and Lesner, Hijmans, and Burton pointed out that necrotic pseudocysts had been discovered in the postoperative period that undoubtedly existed previously and would have been discovered if an exploratory laparotomy had been carried out previously.

Splanchnicectomy, in fact, is considered by most American surgeons from a neurosurgical standpoint; it is meant to relieve pain and is supposed to produce anesthesia of the pancreas without affecting the further course of pancreatitis. Consequently—and Priestley seems to have summarized the opinion generally accepted—intervention on the sympathetic nerves is restricted in the United States to cases in which curative procedures have failed and to cases in which the pancreas is completely destroyed and pain has become permanent. Priestley reports on 12 cases operated upon by the neurosurgeons of the Mayo Clinic; a few were improved for one or two years, others for a longer period of time; none, however, were cured for any longer than five years.

I have personally reviewed the late results in 75 cases of left splanchnicectomy carried out from 1942 to 1953. In all of these 3 cm. of the nerve was resected by a lumbar extraperitoneal approach, after exploration of the pancreas and the biliary passages. The results are as follows:

Mortality, 2

Cases followed up for less than one year, 6

Late results (followed from 1-11 years), 67

Results immediately and persistently satisfactory during the period of observation, 40 cases (60%)

Excellent results, although secondarily only, the patient having presented some transitory symptoms for a certain time that subsequently subsided, 11 cases (16%)

Good results, characterized by the disappearance of painful episodes and the persistence of some functional disturbances, 10 cases (15%)

Total of very favorable results: 61 cases (91%)

Cases perceptibly, although incompletely, improved, 5 (7.5%)

One case was a failure and submitted subsequently to a pancreatectomy (1.5%)

In 31 cases splanchnicectomy was completed by resecting the left semilunar ganglion. This seems to be a favorable element of the surgical treatment of this disease.

To these personal results I may add the observations presented by Kment (3 cures), Moulonguet and Verne (2 complete cures, 1 improvement), and Ciceri (11 successful cases followed for several years).

If one considers the qualitative characteristics of the cure (complete and definitive disappearance of painful episodes, rapid improvement of the general condition of the patient, weight gain of 10 to 20 kg.), one may assume that left splanchnicectomy in the treatment of chronic relapsing pancreatitis is not a symptomatic procedure against pain but definitely a curative procedure, interrupting the evolutional cycle of the disease and putting an end to the continually recurring processes that characterize it.

I may also add that it is worthless to extend the resection of the sympathetic chain above the lumbar segments or to carry it out on the right side also, whereas it is essential to carry out a laparotomy, in order to explore the pancreatic lesions as well as the biliary passages.

Left splanchnicectomy seems to me to be indicated only when operative manometric and roentgenologic studies have established that the sphincter of Oddi is anatomically and functionally normal. If this is not the case, I believe that all attention should be concentrated upon the sphincteric dystonicity.

In cases of hypertonicity a sphincterotomy should be performed according to Doubilet and Mulholland's suggestion (if the spasm is labile, vagotomy should be done).

In cases of hypotonicity right splanchnicectomy should be done, since its elective action on the tonicity of the sphincter of Oddi is well established. In 1950, of 26 cases treated that way, I noted excellent results in 14 cases and good results in 8, in other words satisfactory results in a total of 80% of the cases treated.

In the apparent confusion presently encountered in the surgical treatment of chronic relapsing pancreatitis, it is thus possible to find a lead in the analysis of physiopathologic facts that will permit ascribing precise indications to each type of operation:

Palliative biliary bypass operations whenever manometric and roentgenologic studies have revealed the existence of a compression of the biliary passages.

Right splanchnicectomy or sphincterotomy or even vagotomy whenever there is a dystonicity of the sphincter of Oddi.

Left pancreatectomy whenever the lesions are severe and localized to the left.

Finally, left splanchnicectomy, which with minimal risks ensures proper treatment of chronic pancreatitis, whenever the latter is not caused by a dystonicity of the sphincter of Oddi and whenever a pancreatectomy would be insufficient or not justified.

I have looked up how these various indications are distributed in the practice. Thirty-eight cases of chronic relapsing pancreatitis operated upon during the period of 1952 to 1953 were distributed in the following manner:

Eight cases of internal biliary derivation; in three of these the gall bladder was used and in five the cystic duct. In a total of six cases the procedure was completed by a left splanchnicectomy. In two cases fatal outcome of the disease did not permit the operation.

Eleven right splanchnicectomies and one single vagotomy (there was no definite indication for sphincterotomy).

One left pancreatectomy.

Twenty-three left splanchnicectomies associated with a resection of the left semilunar ganglion.

The late results of these 38 procedures, after a follow-up of one to three years are as follows:

Three deaths (8%).

One patient could not be traced.

Thirty-four late results, all favorable, of which:

26 were excellent immediately (76%)

5 secondarily satisfactory (15%)

5 good results (9%)

Improvement, failure, or recurrence: none.

In conclusion I may say that it is urgent to compare the results obtained on both sides of the Atlantic in the fascinating problem of surgical treatment for chronic relapsing pancreatitis.

It is important to find out why, at a distance of one night's flight, identical operations do not give the same results.

I do believe that all operations suggested have their individual indications, although none of them, even left splanchnicectomy, can pretend to be exclusively satisfactory.